THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—18TH YEAR. SYDNEY, SATURDAY, AUGUST 8, 1931. No. 6.

SPECIAL TUBERCULOSIS NUMBER

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TUBERCULOSIS IN AUSTRALIA.

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The story of tuberculosis in Australia begins with the first recorded burial of a European in New South Wales. On May 1, 1770, Captain Cook landed a burial party at Botany Bay and buried the body of Forby Sutherland.

The following is the entry in Cook's diary:

Tuesday, May 1st, 1770: Last night departed this life Forby Sutherland, seaman, who died of consumtion, and in the a.m. his body was entard ashore at the watering-place. This circumstance occasioned my calling the south point of this bay Sutherland's Point.

Cunningham, Surgeon of the Royal Navy, writing in 1827 an account of the Colony, stated:

The extraordinary healthiness of the climate of New South Wales must be of no trifling importance in the eyes of a European, considering how unhealthy most other countries are . . . on reaching the age of puberty, phthisis is liable to supervene from the rapid sprouting out in stature of our youths at this period: but the European phthisis is uniformly cured, or at least relieved, by a removal hither, if early resorted to.

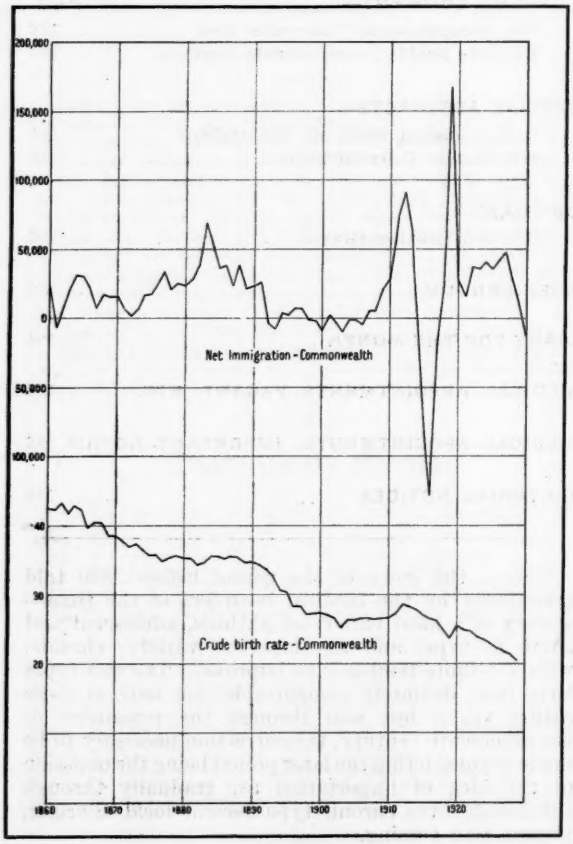
This is the story of the period before 1850 told graphically by the medical recorders of the time—a story of a local variety of phthisis, adolescent and acute in type, and an imported variety, chronic, with a definite tendency to improve. The two types have been definitely recognizable, not only in these earlier years, but also through the remainder of the nineteenth century, the correction necessary to be made in considering the later period being the omission of the idea of importation as, gradually through the decades, the chronic type became local in origin, importation ceasing.

The story of tuberculosis can be told most easily by the presentation of Graphs I, II and III. Graph I shows the net immigration and the crude birth rate of the Commonwealth. It has not been possible to show on this graph the period between 1850 and 1860; but the Commonwealth Statistician gives the following figures of estimated population in the Commonwealth:

1850	405,356
1855	793,260
1860	1,145,585

It is evident from these figures, as it is a familiar historical fact, that the decade 1851–1860 saw the

greatest proportional population increase the Commonwealth has ever known. This was an immigration of young adults: the very abnormally high birth rate seen between 1860 and 1870 was a natural sequence. The next period of immigration movement was between 1880 and 1890. The high birth rate of this period is obvious on Graph I. The third immigration period was between 1908 and 1912 and the rise in the birth rate at this period is equally a notable feature of the birth-rate curve in Graph I. The violent fluctuations in the immigration curve between 1913 and 1919 represent the departure and return of those who were abroad on service during the Great War. The rise in the birth rate in 1920 following the return of the troops is only of minor value.

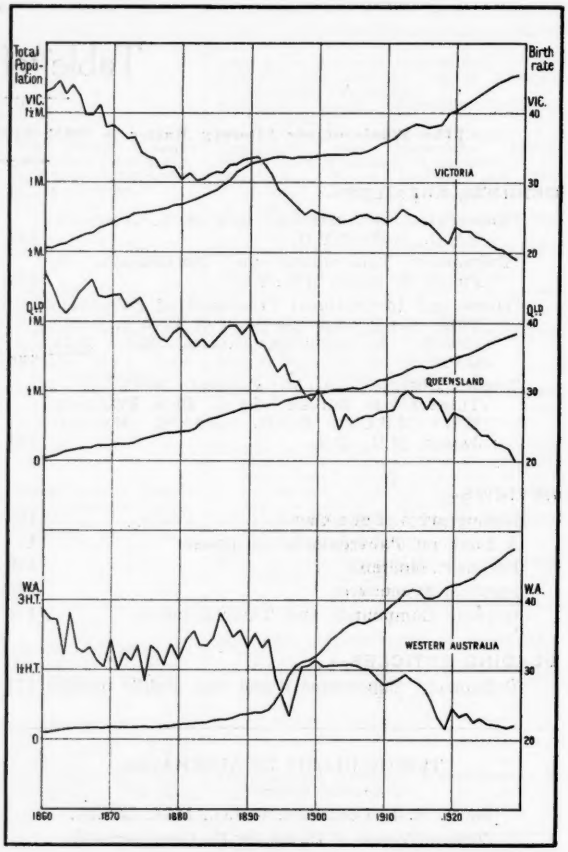


GRAPH I.

Net immigration and crude birth rate for the Commonwealth.

Amongst the six States, three experienced these migration fluctuations in an especial degree. These were the States in which great discoveries of gold were made. Their story is told in Graph II. Victoria had the migration of the 'fifties, but had another migration due to general economic prosperity in the 'eighties. Queensland had a migration wave due to the discovery of gold in the 'eighties, but also due to the great importation of Kanakas to work on the sugar fields. Western Australia had the great immigration of the 'nineties. Each State also showed the minor migration wave around 1910. Graph II shows clearly how each of these waves was followed by a high birth rate.

The infants born at each of these epochs would reach the age of susceptibility to the adolescent type of tuberculosis between twenty and thirty years later.



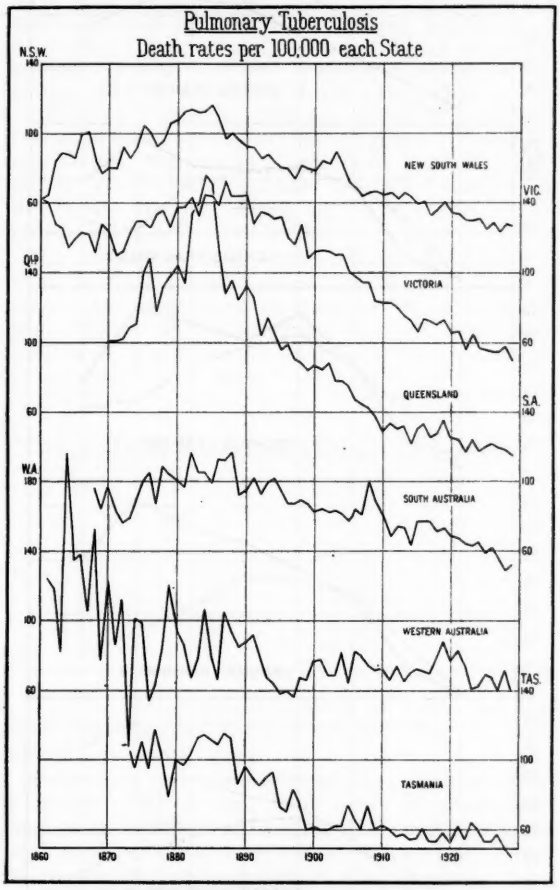
GRAPH II.

Total population and crude birth rate in each of the three States—Victoria Queensland and Western Australia.

Graph III consequently should, by deduction, show an increase in the death rate from pulmonary tuberculosis in each State between 1880 and 1890 and between 1910 and 1920, and the three States in which this should be most apparent should be Victoria, Queensland and Western Australia. Graph III shows that this has actually been the course of the disease. If the method of this deduction is accurate, a slight rise in the death rate from pulmonary tuberculosis is to be expected around 1940, the rise being seen about equally in all States.

Graph III shows one violent departure from the normal course described above; this is seen in the course of the Queensland death rate between 1880 and 1890. In this decade the death rate from pulmonary tuberculosis was phenomenally high. This was due, beyond question, to the very great

numbers of deaths among the Kanakas from an acute type of pulmonary infection, doubtless for the most part ascribed accurately, although probably not invariably so, to tuberculosis.



GRAPH III.
Death rate from pulmonary tuberculosis in each State.

The high levels and abrupt fluctuations in the pulmonary tuberculosis death rate of the nineteenth century have given place, as Graph III shows clearly, to a lower level and more regular course in the death rate. This is consonant with the stabilization of the age-constitution of the population. This is not the place for full discussion of the course of the age-constitution of the population; that has been considered fully elsewhere. Here, however, it may be stated that at the three epochs mentioned—the 'fifties, the 'eighties, and around 1910—the immigration of large numbers of people of young adult age kept the age-constitution of the population at an average level much below that of the standard European population. With the gradual increase in the total numbers of the population the diluting influence of the migrant mass became less until now the population of the Commonwealth has reached an age-constitution approximating very closely to the "standard" age-composition of a stabilized European community.

No further fluctuation in the tuberculosis death rate due to this factor may be expected therefore after 1950; the projected rise about 1940 even is by no means certain.

It is interesting to have contemporary evidence in support of these deductions. In 1876, Thomson, critically examining the course of events in Victoria, stated:

> In briefly recapitulating the immediate inferences deducible from the foregoing figures, it may now be finally affirmed:
> First, that the fatality from phthisis at the ages most liable to the disease has very largely increased.
> Second, that even in that greatly augmented ratio of fatal phthisis among the general population, there has also been a still more remarkably increased proportion of fatal phthisis among the native born.

This report by Thomson evoked controversy and the Medical Society of Victoria appointed a Committee to inquire into the question. This Committee concluded, *inter alia*, that:

> The apparent increase of mortality among young adults is due to the influx of phthisical persons from abroad.

The Government Statist of Victoria replied to this deduction as follows:

> This conclusion is shown to be erroneous by the table. Only three per cent. of the deaths from phthisis in 1876 were of persons who had been in the colony less than one year, whilst 89 per cent. had been there for more than five years, and 81 per cent. more than ten years prior to their death.

I have brought out somewhat prominently this aspect of the course of pulmonary tuberculosis in Australia, as it has been a feature characteristic of this country to a rather especial degree. Other countries have not shown the rapid rise in the tuberculosis death rate seen (Graph III) in New South Wales, Victoria and Queensland between 1870 and 1885. The explanation I have given seems to me the only explanation which is adapted to all the recorded facts.

But it should be stated here, as it will again be stated later, that the adoption of this as one of the factors concerned does not exclude or even obscure the operation of other factors. It does, however, involve this: that the great excess of susceptible adolescents threw the death rate up to a level far higher than should ever have been the normal for a country possessing such notable prophylactic and therapeutic advantages; that we cannot congratulate ourselves overmuch as being agents in the great decline in the death rate in this century; that we must in all humility take the present level of the death rate not as a standard of signal achievement by comparison with the death rates in other countries, but as a natural phenomenon largely independent of our efforts and as, actually, a high rate for a country so happily endowed with all natural advantages and a reproach to all of us, which should be at once removed—a starting line for a race not yet begun.

Victor Heiser, of the Rockefeller Foundation, defined the position when he said on one visit to Australia: "You should in Australia have no tuberculosis."

Comparisons with Other Countries.

And what actually is our position by contrast with other countries?

Representative death rates are shown in the table (Table I).

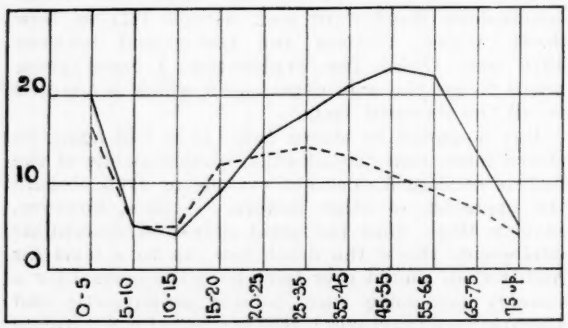
TABLE I.

Tuberculosis Death Rates per 100,000.

Country.	Pulmonary Tuberculosis.	All Forms of Tuberculosis.
New Zealand	41	46
South Africa (whites)	36	51
Australia	47	54
Denmark	61	78
United States (registration area)	71	81
England and Wales	76	93

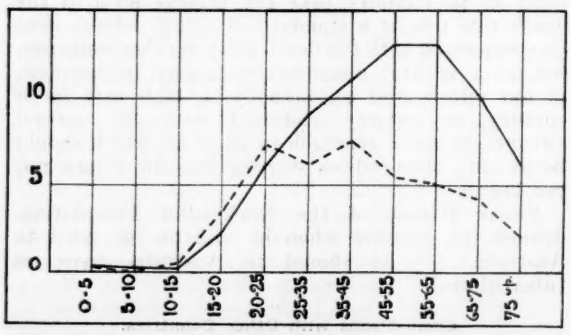
With all the great natural and social hygienic advantages, Australia has a pulmonary tuberculosis death rate more than half that of England with its less favourable climate and much lower economic status.

Graph IV shows the course of evolution of the age-incidence of mortality from pulmonary tuberculosis in Victoria during sixty years. In order to appreciate the significance of this graph, it would be well to study first Graphs V and VI in which is shown the death rate for each age group for England and Wales and for the Commonwealth. In England and Wales there is a high mortality rate in the "under five" age group which is not seen in the Commonwealth. It is hardly practicable to discuss here the full significance of this, but it may be noted at least that Australia has no infantile tuberculosis problem comparable with that of England.



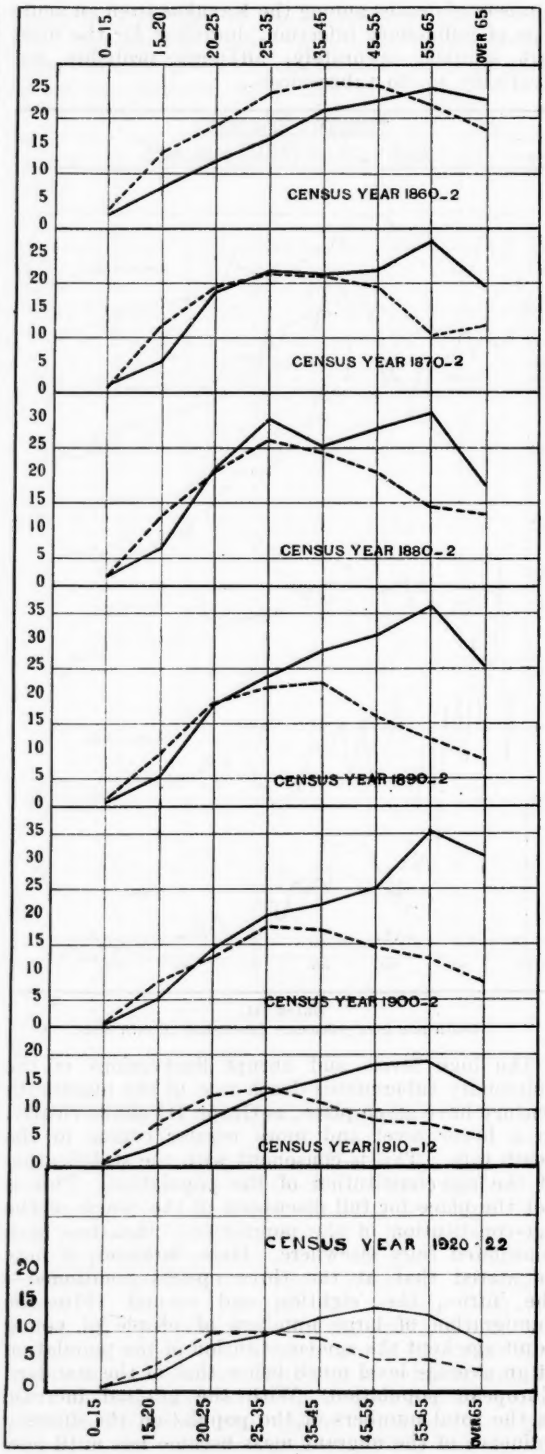
GRAPH V.

England and Wales. Death rates per 10,000 of population according to age distribution—the mean for three years, 1912–1914. The continuous line indicates males, the interrupted line females.



GRAPH VI.

Commonwealth of Australia. Death rates per 10,000 of population according to age distribution—the mean for three years, 1920–1922. The continuous line represents males, the interrupted line females.



GRAPH IV.

Tuberculous in Victoria in census years. Showing death rates per 10,000 of population for each sex in age groups. The continuous line represents males, the interrupted line females.

From five to fifteen the death rate in each country is low; after fifteen it rises steadily with advancing age, reaching its maximum between thirty-five and fifty-five, after which it steadily declines. In these days in Australia, as in England, tuberculosis is a mortal disease of later adult life—at least it is so in males. In females it is fatal mostly to young adults. It is to be noted that these graphs, as also Graph IV, are based on death rates, that is, the number of deaths in each age group in every ten thousand persons living in that age group. Graph IV, representing the course of events in Victoria, brings out in very striking manner the great rise in the death rate amongst young adults as shown by the census years 1870 and 1880.

For comparison, and perhaps representing quantities more familiar to the general practitioner, Table II gives the percentage represented by the deaths from pulmonary tuberculosis in each age group per hundred of the total of deaths from pulmonary tuberculosis at all ages.

TABLE II.

Deaths from Pulmonary Tuberculosis expressed as a Percentage of Total Deaths from All Causes in each Age Group. Commonwealth, All Persons.

Age Group.	Percentage.	Age Group.	Percentage.
0–5	0·7	40–45	10·1
5–10	0·2	45–50	9·9
10–15	0·7	50–55	8·7
15–20	5·0	55–60	6·9
20–25	10·2	60–65	5·7
25–30	11·6	65–70	4·3
30–35	10·7	70–75	2·7
35–40	11·8	Over 75	1·0

This grouping does not differ greatly from that shown by the death rates, the latter being the truest method of presentation.

Pulmonary tuberculosis to its antagonists must be a disease with a firm hold over all adult years increasing the extent of its hold on the male population with each decade of age until at or about the period of senescence cancer begins to take its place.

This is the story of the successful attacks of the bacilli as determined by death—a story, in these days of almost accurate diagnosis, beyond all reasonable doubt. What is known of its attacks on the living? Here there is less certainty.

Penfold, on the results of *post mortem* examinations, reported to the Australasian Medical Congress of 1923 that approximately 25% of males and 22% of females showed definite tuberculous lesions. He commented:

> All the European figures are very much higher than the Australian figures herein recorded, and corroborate the relatively low tuberculosis death rate found in Australia, as against the high rates prevailing in Europe.

The possibility of error arising from birth outside Australia was adequately excluded, and Penfold recorded that:

> The frequency of tuberculous lesions found *post mortem* in Australia is very similar in those born within and those born outside Australia.

Tuberculin tests were done over a wide range of persons presumed to be, clinically, non-tuberculous. The results of these were somewhat various, but on the hospital population 61% gave positive von Pirquet reactions, as did 70% of University students between eighteen and twenty-seven years of age.

These observations should be repeated over a much wider section of the population before any reliable deductions can be drawn. Our present state of knowledge may be indicated briefly by saying that between 60% and 70% of the Australian population are invaded by the tubercle bacillus sufficiently to produce a positive von Pirquet reaction, about 20% to 25% show some tuberculous lesion after death and 0·047% die with pulmonary tuberculosis as the cause of death. The figures must not be accepted as figures; they are, however, fairly reliable as indicators suggesting with a reasonable degree of approximation to the probable truth the rates of invasion, recovery and death in the Australian population.

It should be noted as of great practical importance that here is a very widespread infection strong enough to make for itself a recognizable home in a quarter of the population, but faced with such a strong human resistance that less than one person in every thousand dies. On the one hand a ubiquitous enemy, striking here and there one in every four of us; on the other hand every individual a gallant—and mostly successful—ally in the official war against this disease. These two factors determine the campaign tactics.

The Type of Infection.

But first must be determined the source of the infection—is the disease in Australia human or bovine?

The first important survey of the type of tubercle bacilli found in various types of tuberculous lesions was reported by Penfold to the 1923 Congress.

Of the seventy strains of tubercle bacillus isolated from tuberculosis lesions in adults, all were found to be human in character. These strains were in almost every instance isolated from sputa. In the case of children specimens of material were obtained from sixty patients. Of fourteen specimens of material obtained from the glands of the hilus of the lung, three yielded the bovine strain, that is, 22% bovine. Of thirty-two specimens obtained from children from lesions other than those of the respiratory system and hilus of the lung, seven yielded the bovine strain of organism and twenty-five the human, that is, 22% bovine.

In the case of the mesenteric and portal glands, of ten specimens examined six yielded the bovine type of organism, that is, 60% bovine.

The ages of the patients were carefully recorded. With the exception of one patient of unknown age, all the persons from whom the bovine strain of organism was isolated were under four years of age, and in the first four years of life 26% of the infections were bovine in origin.

An investigation has been carried on at a number of the District Health Laboratories belonging to the Commonwealth Department of Health during the years 1924–1928. In this period tubercle bacilli were isolated and the strain of organism determined

in a series of seventy-eight cases. The source of the material was as follows:

Sputum	71
Pleural fluid	1
Urine	3
Unspecified	3

The material was obtained from adults and in every case the human strain of organism was isolated. This result corresponds very closely with that recorded by Penfold and quoted above.

In Tables III, IV, V and VI are set out the death rates from pulmonary and other forms of tuberculosis in each State and in each capital city. These tables show:

(*a*) That the death rates from pulmonary and from other forms of tuberculosis have decreased in every State and in every capital city.

(*b*) That this decline has been greater for non-pulmonary than for pulmonary tuberculosis and greater in each case in the capital cities than in the States.

(*c*) That Queensland (and Brisbane) show an exceptionally low "non-pulmonary" death rate.

It is necessary to point out, however, that the accuracy of these tables is determined by the accuracy of diagnosis and reliability of the pathological classification for tabulation purposes. There is reason to believe that, in relation at least to "non-pulmonary" deaths the classification has in the past been somewhat unreliable. Table VI shows (especially for Brisbane) about 1885–1886 and again in 1906–1907 a definite change in the order of numerical quantity of the death rates. These were the dates at which the nosological classification was completely changed. This table should therefore be accepted only with reservations.

Holmes, who examined the bovine tuberculosis position for the Federal Health Council, summarized the principal aspects thus:

TABLE III.

DEATHS FROM TUBERCULOSIS. MORTALITY RATES PER 100,000 OF MEAN POPULATION.—STATES OF AUSTRALIA.

Tuberculosis of Respiratory System (Class 31 of International Classification).

Year.	Rate.					
	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.
1871	—	114	—	—	—	—
1872	—	116	—	—	—	—
1873	—	123	—	—	—	—
1874	—	130	—	—	—	—
1875	—	130	—	—	—	—
1876	—	127	—	—	—	—
1877	—	135	—	—	—	—
1878	—	137	—	—	—	—
1879	—	127	—	—	—	—
1880	—	138	—	—	—	—
1881	—	138	135	—	—	—
1882	—	143	174	—	—	—
1883	—	133	180	—	—	—
1884	—	146	196	—	—	—
1885	—	145	192	—	—	—
1886	—	140	152	—	—	—
1887	—	153	129	—	—	—
1888	—	145	137	—	—	—
1889	—	144	136	—	—	—
1890	—	146	133	—	—	—
1891	—	129	126	—	—	—
1892	—	136	104	—	—	—
1893	—	134	115	—	—	—
1894	—	131	105	—	—	—
1895	—	132	96	—	—	—
1896	—	121	99	—	—	—
1897	—	116	91	—	—	—
1898	—	128	90	—	—	—
1899	—	113	85	—	—	—
1900	—	116	87	—	—	—
1901	—	118	85	—	—	—
1902	—	117	90	—	—	—
1903	—	111	78	—	—	—
1904	—	111	79	—	—	—
1905	—	102	75	—	—	—
1906	—	99	67	—	—	—
1907	—	96	63	—	—	—
1908	—	96	62	—	—	—
1909	—	85	58	—	—	—
1910	64	81	51	74	77	68
1911	66	83	58	70	64	67
1912	61	79	53	75	74	62
1913	66	74	54	77	66	59
1914	62	71	44	64	72	47
1915	58	64	50	77	74	53
1916	60	71	54	77	70	59
1917	53	64	46	78	70	48
1918	56	66	48	72	79	46
1919	63	71	56	73	92	57
1920	53	64	46	69	78	50
1921	52	67	44	67	78	61
1922	49	58	39	65	74	48
1923	50	64	44	63	62	63
1924	51	57	37	65	61	60
1925	45	56	40	60	70	51
1926	49	54	42	60	67	51
1927	44	55	40	56	60	57

The incidence of bovine tuberculosis in the community is by no means inconsiderable.

The mortality from human tuberculosis of bovine origin is low.

The mortality from human tuberculosis of bovine origin has been declining very rapidly of recent years, much more rapidly than that from tuberculosis of human origin. This is particularly evident in the cities and may reasonably be ascribed to smaller dosage of infection through the milk supply.

Bovine infection is seldom found except in children under the age of fifteen years and principally under the age of four years.

In a large majority of cases bovine infection appears to be limited to the glands, chiefly the mesenteric, portal and cervical glands. In Australia, laboratory investigations indicate that bone and joint tuberculosis is usually associated with the human strain of bacillus.

The mortality from "non-pulmonary" tuberculosis in the Commonwealth in comparison with that of other countries is indicated by the following death rates:

New Zealand	5 per 100,000
South Africa (whites)	15 ,, ,,
Australia	7 ,, ,,
Denmark	17 ,, ,,
United States	10 ,, ,,
Netherlands	23 ,, ,,
England and Wales	17 ,, ,,

From this table it is seen that the incidence of bovine infection in the Commonwealth compares very favourably with that in other countries.

The distribution of bovine infection amongst bovine animals in Australia has not been ascertained along any general lines. Limited information only is available. This indicates that in Australia "it would be safe to say that approximately 5% of all abattoir cattle are affected with tuberculosis in some degree."

Concerning the true incidence of tuberculosis amongst dairy cattle little is known.

TABLE IV.

DEATHS FROM TUBERCULOSIS. MORTALITY RATES PER 100,000 OF MEAN POPULATION.—STATES OF AUSTRALIA.

Tuberculosis other than of the Respiratory System.

Year.	Rate.					
	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.
1871	—	32	—	—	—	—
1872	—	32	—	—	—	—
1873	—	35	—	—	—	—
1874	—	37	—	—	—	—
1875	—	48	—	—	—	—
1876	—	42	—	—	—	—
1877	—	38	—	—	—	—
1878	—	34	—	—	—	—
1879	—	30	—	—	—	—
1880	—	38	—	—	—	—
1881	—	33	20	—	—	—
1882	—	33	33	—	—	—
1883	—	30	39	—	—	—
1884	—	32	45	—	—	—
1885	—	36	71	—	—	—
1886	—	36	62	—	—	—
1887	—	38	69	—	—	—
1888	—	35	60	—	—	—
1889	—	41	56	—	—	—
1890	—	39	51	—	—	—
1891	—	42	44	—	—	—
1892	—	34	44	—	—	—
1893	—	29	47	—	—	—
1894	—	27	42	—	—	—
1895	—	30	33	—	—	—
1896	—	30	36	—	—	—
1897	—	31	30	—	—	—
1898	—	35	35	—	—	—
1899	—	31	37	—	—	—
1900	—	31	32	—	—	—
1901	—	31	37	—	—	—
1902	—	26	32	—	—	—
1903	—	29	27	—	—	—
1904	—	31	18	—	—	—
1905	—	28	15	—	—	—
1906	—	27	13	—	—	—
1907	—	21	10	—	—	—
1908	—	20	10	—	—	—
1909	—	19	9	—	—	—
1910	11	16	8	23	8	13
1911	10	16	9	15	14	19
1912	10	14	9	13	11	18
1913	12	13	5	13	8	15
1914	9	12	5	10	6	11
1915	8	12	5	14	10	11
1916	9	13	5	15	7	22
1917	7	14	4	15	8	13
1918	8	14	4	10	8	16
1919	8	11	5	13	7	17
1920	9	13	4	11	7	14
1921	7	12	3·8	12	7	10
1922	7	10	2·7	10	8	20
1923	6	12	1·9	8	4	12
1924	5	13	4·7	7	7	16
1925	6	10	2·2	10	8	12
1926	6	8	2·9	10	4	10
1927	5	10	1·9	8	5	11

Tables III to VI show that both pulmonary and non-pulmonary tuberculosis are more fatal in the capital cities than in the rural districts without being exclusively or even disproportionately metropolitan diseases.

Tuberculosis in Immigrants.

It is important that we should know whether any considerable proportion of the total deaths from pulmonary tuberculosis is of persons who have recently arrived in the Commonwealth and may be presumed to have brought the disease with them. This is determined by Table VII.

TABLE VII.

Length of Residence in Australia.	Percentage of Total Deaths from Pulmonary Tuberculosis.
Under five years	2·5
Five years and under ten	2·5
Ten years and under fifteen	1·1
Fifteen years and under twenty	3·1
Twenty years and over	8·2
Born in Australia	78·8
Unspecified	3·2

Table VII relates to the year 1929 and may be amplified as follows: a total of 3,464 persons died in that year from pulmonary tuberculosis; of these, 2,732 or 78·8% were born in Australia while 87 or 2·5% had lived less than five years in Australia, 233 or 6·7% had lived in Australia more than five but less than twenty years, and 286 or 8·2% had lived in Australia twenty years or more. It is impossible from these figures alone to say how many of these migrants brought their tuberculosis to Australia and how many were infected here. Those who had been here less than five years call most for consideration as having possibly been identifiable at the time of arrival. Even with all the other collateral evidence which is available and with the great accumulated experience of the Department, I do not find myself able to form an estimate of how much of this tuberculosis—fatal within five years after arrival in Australia—was imported and how much was detectable at the time of arrival. My general impression, which is not without value, is that a considerable proportion is imported but a very small proportion indeed would have been detectable on arrival even with very rigorous examination. This relates only to the pulmonary tuberculosis fatal within five years after arrival; of the tuberculosis fatal in persons who were not born in Australia at longer intervals after their arrival, nothing can be said with even a slight degree

TABLE V.

PULMONARY TUBERCULOSIS—CLASS 31.

Death Rates per 100,000 *of Mean Population in Certain Cities.*

Year.	Sydney.	Melbourne.	Brisbane.	Perth.	Hobart.	Launceston.
1880	187	—	—	—	—	—
1881	181	227	190	—	—	—
1882	206	231	236	—	178	156
1883	150 } 187	223 } 233	257 } 275	—	192	169
1884	193	244	304	—	157 } 179	214 } 186
1885	203	239	390	—	217	166
1886	172	212	174	—	151	226
1887	162	232	106	—	160	205
1888	168	215	128	—	153	105
1889	141	208	124	—	122	105
1890	148	200	156	—	168	135
1891	143	173	99	—	162	120
1892	123	192	86	—	146	108
1893	117	185	121	—	134	112
1894	116	176	74	—	149	119
1895	100	182	86	—	99	91
1896	98	159	160	—	70	84
1897	88	161	120	—	124	114
1898	107	169	139	111	78	98
1899	101	135	125	102	72	62
1900	95	140	124	101 } 106	90	85
1901	114	155	121	120	104	80
1902	103	143	104	95	78	65
1903	101	140	92	99	86	84
1904	96	135	88	126	74	102
1905	81	122	93	92	115	88
1906	78	115	80	127	80	92
1907	74	116	94	115	86	46
1908	70	115	91	92	91	49
1909	69	97	78	84	73	40
1910	72	97	71	98	59	64
1911	69	99	87	96	92	85
1912	57	100	75	100	89	77
1913	65	88	97	105	88	50
1914	56	89	72	103	56	49
1915	51	77	89	82	88	69
1916	54	86	88	49	87	89
1917	46	79	74	46	60	96
1918	48	83	73	48	53	64
1919	40	87	80	61	82	104
1920	51	79	73	56	65	87
1921	49	81	66	69	96	71
1922	46	67	54	60	77	69
1923	44	69	64	56	98	76
1924	49	65	58	67	104	55
1925	38 } 45	63 } 63	55 } 58	72 } 64	91 } 100	59 } 61
1926	44	59	61	64	98	52
1927	48	63	51	80	108	64
1928	53	—	44	—	100	63

NOTE.—The Military Base Hospital and the Tasmanian Sanatorium for Tuberculosis are in Hobart and deaths in these institutions are included in the Hobart figures.

of certainty as to the relative probabilities of importation or of local infection.

Industrial Occupation.

The relationship between industrial occupation and pulmonary tuberculosis has been under examination from time to time. The mining industry has been well studied. There is hardly sufficient space here to give all the evidence, but some of the principal facts can be indicated.

Taking the census year 1921, the great occupational groups show mortality as follows (Table VIII):

TABLE VIII.

Pulmonary Tuberculosis and Occupation.

Occupational Group.	Male Population.	Number of Deaths.	Mortality Rate per 1,000.
Professional	121,483	167	1·37
Domestic	49,972	95	1·90
Commercial	275,325	292	1·06
Transport and Communication	200,523	165	0·82
Industrial	604,676	784	1·29
Primary Producers	588,310	404	0·69
Total Occupied Males	1,840,289	1,907	1·04

The mean rate for the whole male population is 1·04‰. Transport workers and primary producers —those who work in the open—are lower than the mean; the commercial workers are at the mean level, then come the industrial, professional and domestic, in that order.

This table really does not signify much—more detailed knowledge as to special occupations is essential. For example, we may point out the contrast between railway workers with a rate per thousand of 0·46 and "mining and quarrying" workers with a rate of 3·20‰—more than three times the normal for all males.

So much for the deaths; there is evidence also of the incidence of pulmonary tuberculosis in the living. The morbidity rate amongst the teachers in the Education Department of Victoria was examined by the Industrial Hygiene Division of this Department in relation to the years 1914 and 1920, 1921, 1922. Table IX shows the numbers of verified cases of pulmonary tuberculosis among the teachers.

The mean rate for the four years is 1·5‰.

In the Government Railways of New South Wales and Victoria with 44,506 and 21,474 employees respectively, the rates per thousand of persons

TABLE VI.

TUBERCULOSIS OTHER THAN PULMONARY.

Death Rates per 100,000 *of Mean Population in Certain Cities.*

Year.	Sydney.	Melbourne.	Brisbane.	Perth.	Hobart.	Launceston.
1881	—	58	10	—	—	—
1882	—	59	27	—	36	16
1883	—	52	64	—	80	6
1884	61	54	86	—	31	—
1885	67	58	100	—	13	11
1886	82 } 71	64 } 61	70 } 77	—	16 } 26	—
1887	73	65	81	—	22	26
1888	72	64	49	—	38	29
1889	65	68	54	—	54	60
1890	45	65	68	—	52	51
1891	45	66	35	—	37	37
1892	44	53	36	—	72	68
1893	50	45	34	—	63	40
1894	48	43	38	—	28	44
1895	36	44	29	—	56	69
1896	37	43	49	—	43	34
1897	32	39	35	—	44	37
1898	33	52	44	17	38	39
1899	29	40	64	21 } 22	67	33
1900	18	43	39	14	35	33
1901	19	44	35	28	38	24
1902	24	39	44	28	43	47
1903	20	42	27	25	23	38
1904	22	44	17	19	63	46
1905	19	39	16	15	34	28
1906	17	39	18	18	45	46
1907	21	34	9	22	48	97
1908	20	26	5	28	58	69
1909	17	26	10	24	40	28
1910	17	24	11	20	37	44
1911	13	26	16	20	45	21
1912	13	20	17	20	24	38
1913	17	22	7	14	21	33
1914	13	20	10	12	15	25
1915	11	17	9	14	28	24
1916	11	18	8	11	31	24
1917	9	22	4	11	22	23
1918	11	18	5	12	28	11
1919	7	17	9	13	18	27
1920	11·5	19	5	18	16	23
1921	10·3	19	3	13	16	15
1922	7·7	15	5	15	25	22
1923	7·1 } 7·3	14 } 12	2 } 3	5 } 7·6	24 } 17	14 } 20
1924	5·8	14	6	7	14	29
1925	7·7	11	2	10	17	22
1926	9·5	9	3	8	17	7
1927	6·3	12	2	8	12	28
1928	7·6	—	4	12	21	21

TABLE IX.
Pulmonary Tuberculosis among Teachers.

Year.	Total Number of Teachers.	Infections per 1,000 Teachers.
1914	5,349	1·31
1920	6,206	2·09
1921	6,621	1·06
1922	6,478	1·70

ill with pulmonary tuberculosis in the year 1928 were:

New South Wales	1·1
Victoria	1·5

The close approximation of these three sets of figures is very striking: in three large bodies of representative people the rate of incidence of pulmonary tuberculosis is between 1‰ and 2‰.

More comprehensive information is available in relation to those engaged in mining and quarrying. This chapter in Australian public health research should be studied deliberately, Table X gives a partial picture only.

TABLE X.

Group of Persons.	Persons Examined.	Number with Pulmonary Tuberculosis.	Rate per 1,000.
Miners, Broken Hill ..	6,538	209	32·2
Sandstone workers, Sydney	716	54	75·4
Metal miners, Tasmania ..	652	21	31·1
Miners, Western Australia	4,067	155	38·2

The great disparity between the incidence rate amongst this industrial group and that in the general population is apparent.

Family Infection.

One further aspect of the tuberculosis infection requires to be noticed—what evidence exists of any special incidence of tuberculosis due to family infection or family predisposition or both? This question has not yet been closely studied, but certain inquiries have been made.

In 1904, Robertson and Norris made careful personal investigation into 529 deaths from pulmonary tuberculosis in Victoria. The relative conclusion was "in 30% of the cases where any information was obtainable, a history of a previous case in the family was obtainable."

In 1910, I personally made inquiry into the circumstances associated with 127 notified cases of pulmonary tuberculosis in Western Australia, and a definite history of contact with other members of the family suffering from tuberculosis was obtained in 36 cases or 28·3%.

These two results are strikingly similar.

The histories of 419 persons receiving invalid pensions on account of tuberculosis were examined in 1923 by officers of this Department. Tuberculosis was recorded in other members of the family in 23% of the cases.

With the accumulation of evidence at the tuberculosis dispensaries in the different States will come knowledge of how many persons in the family of an identified pulmonary tuberculosis patient become invaded by tuberculous infection or clinically declared as tuberculosis cases. This will provide another link in the chain of knowledge.

General Conclusions.

It is now possible to review the tuberculosis problem and, the subject being so large, certain broad aspects are all that can be indicated.

In Australia bovine tuberculosis has been in the past more than a minor factor in morbidity and mortality, as a factor in mortality it is now of minor importance, it is declining as a morbific force, and now bovine infection is seldom found except in children under fifteen years and principally under the age of four years. Public opinion and administrative initiative are alternately pushing each other along the right road and, although we may be impatient at the rate of progress, we know as yet of no shorter road than that we are travelling and we can hope for no more than to be able by this means or that to increase the speed of the machine.

Pulmonary tuberculosis is human in origin—the decree may almost be made absolute; for all purposes of control it is the golden rule. In the industrial field one group of workers is exposed to a very special risk; but, legislatively and administratively, this risk is now provided for, at least in Australia, to the very limit of human knowledge. All else, the bovine infections and the silicosis casualties excluded, becomes a problem expressible in simple terms. There is constantly occurring the invasion along a known lymphatic route by a known bacillus of a fairly constant proportion of the people of Australia. In every thousand people (subject to the declared limitations of numerical indicators and to the incompleteness of our knowledge) some six hundred or seven hundred are invaded by the tubercle bacillus; of these some two hundred show visible lesions after death, fifteen become clinically identifiable as infected, and 0·5 die from pulmonary tuberculosis.

This calls up somewhat sharply the picture of the other infectious diseases. We have become familiar with the feel of an epidemic, whether it be of diphtheria, scarlet fever, or of the central nervous system infections. In each case a large number of people is invaded by the bacillus mostly with mild vague and unrecognizable symptoms, some develop barely identifiable mild attacks, some become fully declared "cases" with text-book attacks, and a few die.

Until we link tuberculosis with the acute infectious diseases, we will not individually have put ourselves in correct posture for attack.

But there is a practical difference. In the acute infections our first working question is: "Whence did this infection come?" In pulmonary tuberculosis our first question must be: "Whether has this infection gone—is going?"

When we have discharged our duty to the patient, put him on the road to non-infectivity and perhaps personal recovery, then immediately, and before leaving the house, the question should be asked:

"Who has been within range of this infection near enough and long enough to come under inspection?" And promptly such inspection should begin—repeated tuberculin tests of each member of the family and the most critical scrutiny of each positive reactor till all reasonable doubt of his perfect health has, years later, been removed. The task is to prevent any of the six hundred joining the two hundred and any of the two hundred joining the fifteen, and for this all aspects of immunity and resistance should be considered and appropriate action taken. At present the vision of a successful tuberculosis campaign is centred rather too narrowly on the declared case. No reason can be advanced for remitting vigilance in this direction, but vigorous action to prevent the person exposed to infection from becoming infected and then infective will prove very profitable ten or fifteen years hence.

The other phases of pulmonary tuberculosis and its control have been deliberately omitted from this discussion; it was considered that the most profitable use which could be made of the available space was to emphasize that aspect which I consider to be by far the most important.

PULMONARY TUBERCULOSIS AND NOTIFICATION.

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SINCE the modern organized campaign against infectious diseases was set in motion towards the end of last century, all text books on preventive medicine have placed notification of infectious diseases in the front rank of the forces attacking the hosts of infection. When, therefore, the discovery of the tubercle bacillus in 1882 settled the much-debated question of the infectiousness of *phthisis pulmonatis*, it was natural that efforts should be made to include pulmonary tuberculosis amongst notifiable diseases. Opposition to this step arose chiefly from the social stigma supposed to be attached to those affected with the disease, and the fear of concealment of cases. During the last decade of last century the agitation for compulsory notification gained force. The feeling amongst the medical profession in South Australia was so strong that, after the *Public Health Act* of 1898 had been introduced into Parliament, they secured the inclusion of special clauses legalizing the compulsory notification of pulmonary tuberculosis by the medical practitioner attending on or consulted by such a patient. On the principle that half a loaf was better than no bread, they agreed to make this notification without fee. They have been allowed to continue on this ration for the thirty-two years since, and should be thankful, perhaps, that no attempt has been made to reduce the standard of living. The other States followed suit at various dates, but in all of them the usual fee for notification is paid to the medical practitioner. New South Wales made pulmonary tuberculosis notifiable in Sydney in 1904, in certain surrounding proclaimed areas in 1915. But the practice was not extended to the rest of the State until March 1, 1929. Victoria introduced notification of pulmonary tuberculosis in 1909 and of all forms of tuberculosis in 1920. Notification of pulmonary tuberculosis became law in Queensland in 1904, in Tasmania in 1919, and in Western Australia in 1912. In the latter State medical practitioners also have to forward an annual return of all deaths from any form of tuberculosis attended by them.

In 1911 a Conference of Principal Medical Officers for uniform measures for the control of consumption in the States of Australia resolved among other things that:

> The indispensable foundation of every scheme for the prevention of phthisis is compulsory notification of all cases of the disease. Notification must therefore be universal, and the form in which cases are notified be uniform throughout Australia. Foremost in any scheme of control must be placed compulsory notification. Such notification must be obligatory, uniform, and universal.

We have waited twenty years, and we are still far from realizing this ideal, or there is still considerable variation in the standards of notification in the different States. But this is the customary attitude in regard to tuberculosis. We have campaigns against foreign invaders like small pox and plague, but like the poor, "tuberculosis ye have always with you." In those twenty years, we have had inter-state conferences on small pox, plague, influenza, venereal diseases, cancer, poliomyelitis, industrial welfare, school hygiene, sanitary engineering, standardization of food and drugs, and statistics, but never one on tuberculosis. Yet in spite of its falling death rate, pulmonary tuberculosis is still one of the greatest causes of death in the Commonwealth. All forms of tuberculosis still cause over 3,000 deaths in Australia each year.

Now, after twenty years, many practitioners are again questioning the value of notification in this disease. The spectre of publicity and its dangers has vanished. But more alarming is the doubt as to its usefulness. The foe is retreating, but the force of facts compels the belief that notification, as hitherto practised, has played but a minor part in causing the retreat.

It is argued that if the weapon were powerful, its introduction should be reflected in the death rate from pulmonary tuberculosis. But a comparison of the falling death rate in different States shows no such alteration in the rate of the downward fall consequent on notification being placed on the Statute Book. On the other hand, a comparison of the tuberculosis death rate in South Australia with that of Australia as a whole shows that, whereas before 1905 the rate in South Australia had been below that of Australia as a whole, about that time it rose above the rate of the whole Commonwealth and has remained higher since. And this in spite of the fact that South Australia had introduced notification in 1899—the other States only some years later.

Various reasons are given for this apparent failure. It is said that practitioners fail to notify cases till too late, often not until they send in the death certificate. A comparison of the number of cases notified in different States each year with the number of deaths from the disease bears this out. But in

that respect figures show that the same thing holds good both for America and England.

This weakness is not necessarily due to deliberate concealment. There is some ambiguity about the wording of the different Acts. In some States the responsibility is on the practitioner attending; in others on the practitioner examining. The position also is different from the acute infection like measles or scarlet fever in which the diagnosis is made within the first few days. The practitioner may suspect the presence of active tuberculosis in his patient because of general symptoms, but at the beginning of his attendance be unable to prove it by local signs or examination of sputum. If he continues to attend, he may overlook the fact that he has not notified; if subsequent attendance falls to another practitioner, the latter may think notification has been made by the former medical attendant. From the wording of the Acts it would follow that, if strictly complied with, every examiner for invalid pensions would notify many cases, with useless duplication; on the other hand, less strict compliance with the Act necessarily leaves gaps in the number of cases notified early.

Often, too, in old people the practitioner has his fancy so set on the old favourite "chronic bronchitis" that he overlooks pulmonary tuberculosis as a possible starter till the colours are up—or even the patient's number is up. Whatever the reason, there is no doubt that in many open cases notification is not made until many contacts have been infected. The remedy, of course, is earlier and more accurate diagnosis. But this leads to other difficulties which will be discussed later.

More important is the fact that notification of any infection is of value only if followed by administrative methods. In the acute infections these measures are chiefly directed to the discovery of the source of infection and to the isolation of the patient while infective. But from the very life-history of the patient with pulmonary tuberculosis both these courses are inapplicable. In only a small proportion of cases can recent massive infection be traced; in the majority of cases it is years since the infection was contracted; some recent illness or privation has lit it up again, and made it an "open" case, and therefore infective. From the chronicity of the disease also, isolation of patients is impossible; nor is it generally necessary. Each of us can point to patients who have lived for years in the company of others without spreading infection because of their scrupulous care in their personal hygiene. The tuberculous patient is more in the nature of an intermittent carrier, and measures of control should be directed to that end.

The appropriate measures of control are outside the scope of this article. It is merely necessary to say that the consensus of modern opinion is that there needs to be a definite scheme of central control in each State with a responsible medical officer in charge, and that the central pivot of the scheme is some form of clinic for tuberculosis, not for treatment but with ample facilities for diagnosis to aid medical practitioners in early diagnosis of suspected cases and contacts. Such clinics are preferably worked in conjunction with general hospitals, under a special medical officer, who is linked up with the State Director of Tuberculosis.

As stated in the report of the 1911 Conference, "compulsory notification is justifiable only when the information thus obtained can be utilized by an organization adequately equipped and competent to carry out the subsequent action which notification exacts. The necessary measures which must follow if compulsory notification is to be effective and of administrative value, must provide for individual as well as public prophylaxis."

No State in Australia has yet succeeded in building up such an organization. In two States the framework has been erected, but the building is not completed; in the others there is but a mass of unrelated structures and loosely assembled materials scattered about the field.

Recognizing these facts, Dr. M. J. Holmes, in his very complete report on "The Control of Tuberculosis in Australia," which was published in 1929, includes the following in his recommendations for future action:

(1) Early notification is essential to prevention in order that infective cases may be placed under suitable supervision and control before they have spread the infection. Consequently, every effort should be made to ensure the earliest possible notification, and as this depends primarily on the co-operation of the general practitioners, no action should be neglected which will bring the general practitioner into sympathy with the system of control. Close personal touch between the medical officer in charge of the clinic and the medical profession in his district is important, together with the extension to the medical practitioner of every possible facility for diagnosis and assistance in treatment. Action through medical officers of health to facilitate collection and laboratory examination of sputum and to induce the medical practitioner to notify early is desirable.

Where a definite scheme of control is in operation in a State, the scheme might be placed fully before the State Branch of the British Medical Association with a request that medical practitioners be circularized and asked to co-operate and to see that all facilities offered for diagnosis are availed of, and that notification is made at the earliest possible moment.

(2) 1. Notification of Cases.—In view of the dependence of the control system upon notification of cases, State Governments are urged to make the necessary amendments to legislation as early as possible, in order to give effect to that portion of Resolution No. 11 of the second session of the Federal Health Council, March, 1928, recommending that all forms of tuberculosis should be made notifiable in all portions of the States.

It is recommended that the Form of Notification should include information as to:

(*a*) Whether the disease is pulmonary or non-pulmonary;
(*b*) the site of the lesion.

2. Where legislation does not already exist for the purpose, legislation should be enacted to provide that notification should be made by the medical man who examines a person who is suffering from tuberculosis or from any sickness, the symptoms of which raise a reasonable suspicion that it may be tuberculosis.

3. Cases of tuberculosis should be notified direct to the State Health Department as well as to the local health authority.

4. Notification of Deaths.—Deaths from tuberculosis should be notified direct to the State Health Department.

5. Fees for Notification.—Fees for notification should be paid automatically. A doctor notifying a case should not be required to forward an application for the notification fee.

These recommendations mark a definite advance on the futilities of our present methods, but they avoid meeting the two main difficulties in the way of advance.

The first is the question of early diagnosis. Every one rightly insists on the importance of this. But

the object of early diagnosis in the acute infections is to prevent an already infective individual remaining a menace to others; the object of early diagnosis in tuberculosis is, if possible, to prevent the individual becoming infective and becoming a menace to others. When we talk of notifying a person "who is suffering from any sickness, the symptoms of which raise a reasonable suspicion that it may be tuberculosis," are we intended to notify non-infectious cases? The majority of young adults suffering from a so-called "primary" pleurisy with effusion, for instance, may be reasonably suspected to be suffering from tuberculosis, especially if inoculation of the fluid into a guinea-pig and a Mantoux or von Pirquet test proves positive. But they are non-infectious and our hope is to keep them non-infectious.

A child who has been in close family contact with an advanced case of pulmonary tuberculosis and who shows loss of weight, loss of strength and recurring slight rises of temperature may reasonably be suspected to be suffering from tuberculosis and this may receive confirmation by radiological investigation. But such a person is non-infective and with proper supervision may remain non-infective. We desire the supervision; do we require notification to get the supervision? Or are we sure of the necessary supervision even with notification?

Most, if not all, the present talk about early notification is so much waste of breath, because it misses this point. If by this phrase we mean the notification of non-infectious cases, we are introducing a new principle in infectious disease, with the exception of tertiary syphilis (under the *Venereal Diseases Acts*) and some cases of leprosy (with our present methods). But recent movements to correct these anomalies are adverse arguments to the adoption of this new principle in tuberculosis. This does not necessarily mean that it is a wrong principle; it might perhaps be justified on the same grounds as the notification of cases of industrial poisoning. But all our present legislation in regard to infections presupposes notification to apply only to patients that are infectious. If we are to extend notification to all cases of tuberculosis—to tuberculous meningitis, acute miliary tuberculosis, pleural tuberculosis, tuberculous adenitis, early tuberculous joints—even though they are non-infective, then let it be stated openly. To the patient himself there would seem to be no great advantages in such a course being followed, in such instances as acute miliary tuberculosis or tuberculous meningitis. Would there be a gain to the community from notifying these and other non-infective forms of tuberculosis?

In order to test the question, it would appear quite an interesting and serviceable experiment to make acute pleurisy notifiable in a particular area for a period and watch the results. For clinical experience shows that pleurisy with effusion, if subsequent treatment of the convalescent patient is neglected, is frequently followed within two years by active pulmonary tuberculosis. If notification of the pleurisy entailed supervision after convalescence, this development might be prevented. Similarly to compel notification of dry pleurisy where it is neither traumatic nor a complication, say, of pneumonia or lung hydatid, would deepen the practitioner's sense of responsibility and prevent the too frequent diagnosis of this condition on the strength of a pain in the side and a doubtful friction rub. We might then train ourselves to regard bodily wasting in a child who is a familial contact with the same seriousness as we regard gluteal wasting in a suspected tuberculous hip. We might even come to regard effusion into the serous cavity of the pleura as important in a suspected tuberculous lung, as we regard effusion into the synovial cavity in a suspected tuberculous joint.

On the other hand, just as we have reached this standpoint in regard to joints without the aid of notification, could we not do the same in regard to lungs and glands? Such a solution of the problem is being attempted in the Federal Capital Territory under regulations which compel medical practitioners to notify infective cases, draws their attention to the necessity of supervision of the undersized or under-nourished child, especially if a family contact, places the resources of the hospital at the practitioner's disposal for radiological or serological aids to the diagnosis of underlying tuberculous infection, and instructs the Medical Superintendent to inform the medical practitioner of the results of such investigation. The working of these regulations will be followed with interest elsewhere.

The other point is the destination of the notification. In most States at present this is the local authority; in some it is also the head of the State Health Department. The recommendations of Dr. Holmes would make the latter practice uniform throughout Australia. Presumably the medical practitioner would in justice be paid a double fee for the double notification; in that case the public has a right to demand increasedly efficient control as the result. Will it get this?

In the light of past experience it is doubtful whether the local authority is the best body to deal with pulmonary tuberculosis. Unlike most of the acute infections, the cause of the illness is quite often not in the area controlled by that particular local authority; if it is, it is more often an economic defect in the patient's circumstances than a sanitary defect in his surroundings. The chronicity of the complaint, and the patient's nomadic habits caused by his search for health or suitable occupation, frequently result in his passing successively into the territories of several local authorities, so that supervision is intermittent. Hence the suggestion that he should notify the new local authority of his change of address. But how many patients know when they pass the boundary of their old local authority? On the other hand, experience in the attempts to control venereal disease has made it doubtful whether notification to one central authority, in the larger States at any rate, does not lead to congestion of records and delay in action.

A distinction must be made between the average country local authority and the more highly organized body in large municipalities, but on the whole it is true that the local authority has neither the money nor the machinery to provide a tuberculosis clinic. Nor as a rule does it bear any relation to a clinic that may be in existence. The chief powers and

functions exercised by the local authority, especially in the country, relate to sanitation of soil and premises, to pure food supplies (including water and milk) and to disinfection. But it is the economic question which is of paramount importance in the tuberculous individual, the reconciliation of the ideal with the practicable. This involves individual adjustments between the patient's medical attendant and the medical officer of health. But the local authority, being a lay mind, tends to the stereotyped. I have had personal experience of the local authority insisting that all notifications must go through and be dealt with by the lay secretary. It is evident that in cities the medical attendant needs to be in close contact with the medical officer of the clinic. If this officer is in touch with the Director of Tuberculosis, there is a direct line from patient or contact through medical attendant and officer of the clinic to the central authority.

It is evident also that in scattered country districts the chief responsibility for examination and supervision of family contacts must rest with the medical practitioner, and that for this purpose he should be in direct relation with the director—again through the district medical officer if such exists for that particular district.

It is evident also that this medical attendant, rather than a nurse or health inspector, is the only one capable of saying how far a patient is carrying out instructions and whether he is a menace to others. Again I have had experience, more than once, of having a nursing inspector ring up at intervals to know whether the notified patient could not be taken off the infective list because he was looking fat and well, although tubercle bacilli could still be demonstrated in the sputum.

Taking all these points into consideration, if fresh legal authority is sought for notification, would it not be better to alter our methods completely and compel the medical practitioner to notify direct to the district medical officer for transmission to the tuberculosis director, or, in the absence of a medical officer, direct to the director? A form could be drawn up containing particulars whether the medical attendant has recommended to the patient, sanatorium, hospital or home treatment, the disinfection measures ordered, the names of family contacts, their state of nourishment, reaction to intradermal tests, and a request, if necessary, for diagnostic facilities at a clinic or so on. This is only an extension of the principle already begun in New South Wales where the medical attendant is allowed to request an exemption of the household from official visitation if he has given instructions for disinfection of sputum and personal care by contacts. The Victorian Act also allows the medical attendant to instruct as to precautions to be taken. The further recognition of the possibilities of preventive work being done by the medical attendant would lighten the administrative burden. It would also lighten administrative costs, for many contacts could have these investigations done privately if the attendant's report were accepted by the medical officer. If segregation is to be legalized, some such step must be taken to avoid friction between medical attendant and lay authority. And these results could all be obtained by some such system of informative notification to the district medical officer or State Director. It is certain that closer cooperation between medical practitioners and medical administrators along these lines is the next requisite for an effective system of notification.

HOME AND INSTITUTIONAL TREATMENT OF PULMONARY TUBERCULOSIS FROM THE POINT OF VIEW OF PUBLIC HEALTH.

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PULMONARY tuberculosis concerns the public health because of the risk of sufferers from this disease infecting other members of the community. Infection occurs almost entirely through tubercle bacilli present in the sputum. Other sources, fæces and pus, are practically negligible. Hence from the point of view of preventive medicine the problem narrows itself to prevention of infection from sputum.

At first sight this might seem simple, but the problem becomes more complicated, when it is recognized that, unlike other infectious diseases, pulmonary tuberculosis does not run a definite course during which the sufferer can be isolated, but may persist for many years with longer or shorter remissions. During the whole course of the disease sputum may be scant or absent, on the other hand it may be profuse. When present it may contain tubercle bacilli throughout, for a few days only, or may exhibit infinite variation in the relative tubercle free and present phases. The presence of bacilli in the sputum depends on the breaking down of active foci; only while this is going on are they found. There is nothing in the clinical condition of the patient to betray their presence, which can only be determined by bacteriological examination. The sputum of apparently perfectly healthy individuals may be loaded with tubercle bacilli, the first evidence of whose presence may be signs of infection in some other member of the family.

In the course of the disease in every sufferer from pulmonary tuberculosis there exists a stage of longer or shorter duration before bacilli appear in the sputum. The ideal at which we should aim is early diagnosis and treatment before bacilli appear. The accomplishment of this ideal will go far towards eradicating the disease.

Pulmonary tuberculosis is diagnosable and should be diagnosed before tubercle bacilli appear in the sputum.

To effect this it is necessary that the general practitioner should be fully seized with the importance of early diagnosis and should be trained to recognize the early symptoms and signs of the disease. By the time the grosser signs on which we were wont to base our diagnosis appear the battle is half lost.

Unexplained debility, slight pyrexia, loss of weight, dyspepsia, an influenzal attack apart from an epidemic, slight cough explained so often as due to catarrh or cigarettes, unexplained pleurisy or

spitting of blood should arouse suspicion. So, too, should slight differences in movement of the two sides of the chest, slight difference in breath sounds or the presence of a few post-tussic râles.

In every suspected case, whether physical signs are detected or not, radioscopic examination of the chest and bacteriological examination of any sputum present are essential. These two methods of examination should be easily and promptly available to all practitioners free of cost as part of the machinery protecting public health.

In large towns this is practicable. In the country the position is more difficult; but, if we are to reach our ideal, the prevention of open tuberculosis, facilities must be provided for taking reliable radiographic films and examining the sputum of all suspects. The interpretation of these films should be undertaken only by those who have had considerable experience in determining not only the presence, but also the degree of activity of a tuberculous lesion. Many mistakes are made on the subject of activity. An opinion should be given only after studying the films in conjunction with the history, physical signs and symptoms. As regards sputum it must be emphasized that a negative result on bacteriological examination proves only that no tubercle bacilli were found in the specimen examined, not that the patient is free from tuberculous infection. Too often the failure to find tubercle bacilli after one or two examinations lulls to a false sense of security.

It is doubtful whether the general practitioner or the health authority fully grasps yet the paramount importance of early diagnosis and treatment.

The diagnosis made, three alternatives present themselves. The patient may be sent to a sanatorium, to the country for a holiday, or may be allowed to remain at home under supervision.

For the majority of patients the first of these alternatives is the best, at least in theory. A sanatorium should offer him the benefit of change of climate, of good food, a thorough training in the details of treatment by autoinoculation and any special therapy in vogue at the moment. It should also thoroughly drill him in the precautions necessary to be taken to prevent relapse and to avoid him becoming a danger to others. In really early cases three months residence should suffice. The patient can then return to work under medical supervision unless such work is obviously prejudicial.

In practice it is doubtful whether the sanatorium has fulfilled the expectations formed of it. At the inception of the idea of sanatorium treatment it was recognized that a sanatorium to be of use must restrict admission to early cases, and must be staffed by capable, enthusiastic resident medical officers who give each patient constant personal supervision and direction.

Unfortunately, it has been found impossible to restrict admission to patients in an early stage. The patient in the really early stage does not see the necessity for sacrificing time and money and of possibly losing his job in extinguishing the incipient conflagration, nor frequently does his medical attendant. Neither seems to grasp the fact that a month or two sacrificed at this stage spells arrest while a little later the period required for cure grows from months to years, or the chance of cure has been for ever lost. It might almost be said, pulmonary tuberculosis is a very serious malady mainly because it is not recognized to be so in its early stages.

Other factors militating against the efficiency of sanatoria are the practice of filling the beds with patients in the more advanced stages who are allowed to occupy them for many months or even years to the exclusion of more suitable early cases; and of dispensing with resident medical officers from motives of economy. When a medical officer is in charge, too often he is himself a tuberculous invalid incapable of much driving force, or he is the next for promotion in the Health Service without any special aptitude, qualification or training for the position.

No sanatorium of more than a few beds can be efficiently conducted by the daily visits for a few hours of a non-resident medical officer. It takes long to reach the individual inside the patient, to gain his confidence, to grasp his character and all the ramifications of his economic and domestic position, to train him in the details of autoinoculation by rest and exercise, and to drill him efficiently in the essentials which will render him innocuous to his fellows. Not only is a resident medical officer a necessary part of the equipment of a sanatorium, but on his character and ability will depend the success or failure of the institution. Dr. Varier Jones, who at Papworth conducts one of the few unsuccessful village settlements, told me that he declines to employ a medical officer who has had tuberculosis on the ground that he lacks the necessary driving power.

The usefulness of sanatoria would be much enhanced if greater efforts were made to attach to them farms or occupational colonies where suitable arrested cases could be taught the rudiments of a healthier occupation than the one in which they fell sick. This annex to the sanatorium should be educational in function, not a village settlement. Given efficient equipment and management, a stay in a sanatorium is undoubtedly the best treatment for the early case.

Where this is not possible, the patient may be sent to the country, provided he is fully instructed in the details of treatment and provided care is taken to insure that the place to which he goes is suitable as regards aspect, climate, food, housing and surroundings. Many patients are sent away without adequate inquiry to surroundings much worse than those they leave, or without adequate instruction in prevention, and so become disseminators of disease.

The question of accommodation for these patients is from the view of the public health both urgent and difficult. Unfortunately, the general public and many nurses have reached a stage of half education in which they see danger in contact with any consumptive. For them the bacilli are ready to spring from the patient's skin and to fly in his breath. They fear to live even next door to him.

The problem would be greatly simplified if the fact could be driven home that infection is conveyed only by sputum, and then only by gross carelessness, that the careful consumptive carries no menace to

anyone, that the use of spitting flask or paper handkerchief is a safety not danger signal. Under existing conditions the tuberculous patient is forced in self-protection to conceal his disease and to neglect the simple precautions that render him harmless.

Were a law enforced that no one, whatever the cause, should spit anywhere except into a special receptacle or into a paper handkerchief carried in special containers to be subsequently destroyed, invidious distinction would be lifted from the consumptive and incidentally the occurrence of other respiratory diseases would be lessened. Such compulsion may sound fantastic, but so did ordinary sanitation a hundred years ago, and so but a year or two back did prosecution for littering the streets with tram tickets. In any case the custom of treasuring nasal or bronchial excretion in dainty silk or linen pocket handkerchiefs till disposed of by the unfortunate laundress has little to commend it.

In addition to a sputum container consumptives should carry their own pillow when travelling.

Risk of infection through table utensils seems to me to be slight, except possibly in patients in a very advanced stage, and certainly ceases where such utensils are carefully washed and rinsed in running water. Saliva rarely contains tubercle bacilli, and active involvement of the tongue or pharynx is rare apart from advanced disease.

If allowed and encouraged to take simple ordinary precautions, a consumptive can live with non-infected people in home or hostel without risk to anyone. If through public prejudice he is not allowed to adopt these precautions without detriment to himself, it becomes the duty of the State to provide in each town boarding-houses, or quarters in existing boarding-houses and hotels, where he can reside in peace and comfort without risk to others or annoyance to himself.

Public ignorance and prejudice on these points are important factors in the spread of tuberculosis.

In deciding whether instead of going to a sanatorium or the country a consumptive in an early stage may remain at home under supervision we should be guided by the following rules: Only those patients should stay at home who really cannot get away; the curative value of change to a suitable climate and surroundings can scarcely be overestimated. Only those should stay whose home surroundings are good and when obvious improvements can be made in the patients' mode of living; only when the medical attendant is trained and prepared to supervise the patient's life conscientiously and when he is sure that valuable time is not being lost. On one hand one is often struck by how little alteration in habits and environment is required to tip the scale in favour of the patient; on the other, not infrequently one meets patients whose chance of cure has been lost through their medical attendant allowing them to remain at home, either from fear of offending or losing the patient, or from ignorance of the importance of early sacrifice.

This raises the question should the general practitioner look after such patients or should he transfer them to a so-called specialist. Often the general practitioner is ignorant of the fundamental principles and rules of treatment, sometimes he is careless and frequently, like the patient, he has blind faith in the value of drugs. At times he is loath to lose a patient and let him go to the care of others. On the other hand he has opportunities denied the specialist of knowing his patient's character, means, domestic surroundings and obligations and, if trained and conscientious, can do everything that is needed at least as well as the latter.

The medical student in the course of his training gets much instruction in such specialties as ophthalmology, rhinology, orthopædics, urology and psychiatry, but little or no practical teaching in the early diagnosis and home treatment of tuberculosis, a disease where life depends often on his knowledge and conscience.

An important step in the eradication of tuberculosis is the training of every practitioner not only in early diagnosis but in the practical details of treatment. Unfortunately, such teaching cannot well be undertaken in a dispensary. Dispensary patients resent students. When the student comes, the patient ceases to do so.

In deciding the line of treatment to be adopted, a broad view must be taken of all factors, including personal, domestic and economic. The economic factor is most often the deciding one, the patient's financial resources determining the plan to be adopted.

For the poor the anti-tuberculosis dispensary offers the best solution and is probably our most important weapon in treatment and prevention. It detects the early case, goes to the home controlling the life of the patient and his people, secures the examination of contacts, teaches sane living, carries out such special treatment as may be suitable, directs the patient to a sanatorium or the country, and watches him on his return.

Anti-tuberculosis dispensaries should be widely established, but only in connexion with general hospitals where advantage can be taken of the general beds, special departments, radiographic, rhinological, pathological, and others whose aid may be needed in the elucidation of obscure cases. To the dispensary should be sent every tuberculous patient leaving hospital. The dispensary should comprise a specially trained whole-time medical officer who should be responsible for organization and, if necessary, district visiting; one or more trained nurses and one or more supervising honorary medical officers. In addition to being open during the day, it should be open on at least one evening during the week.

Conscientious and sympathetic work by a network of dispensaries situate in areas selected to prevent overlapping will do much to insure efficient treatment and prevention. Something has already been done in this direction, though much remains undone.

After many years' connexion with the anti-tuberculosis dispensary at the Royal Prince Alfred Hospital, I feel that the main defects of such institutions arise from frequently changing the resident medical officer in charge, allotting him other duties which prevent him giving undivided attention to the work, the difficulty of regaining touch with patients sent to sanatoria or the country, the difficulty in providing necessities and comforts

for patients without undue trouble and delay; the difficulty of getting contacts away for holidays and into suitable surroundings. Failure on this point allows the incipient and easily arrested case to reach a much more resistant stage before any attempt at arrest is made.

To coordinate the work and to care permanently for the tuberculous population there has been instituted in some States the anti-tuberculosis bureau with a Director in charge.

In theory the bureau controls the tuberculous population of the State, supervises the dispensary, the public or private sanatorium for early or advanced cases, the homes for the dying, the farm colony or village settlement. In practice it largely fails owing to its lack of executive power.

To be of value the bureau must have the right to control the work of the varying institutions, to investigate, classify and direct the work of each and to organize the whole effort.

This can be effected only by direct government control and is then doomed to failure if it is not recognized that the Director must have exceptional knowledge of the disease, unusual organizing capacity and a wide outlook. Such a post must be divorced from departmental succession and must not be at the mercy of the next for promotion to be thrown in turn to someone lower on the departmental ladder when a more lucrative post falls vacant in the service.

Efficiently administered and staffed and given sufficient power, the combination director, bureau, dispensaries, sanatoria and homes for varying stages, with attached farm and training centres should insure at the same time the most efficient treatment for the patient and protection for the public.

A very difficult problem is that of the patient sufficiently recovered to be able to do part-time work, or full-time work for short periods, but in whom continuous full-time work precipitates a breakdown.

To meet this difficulty village settlements have been instituted with varying success. In England, a few in enthusiastic hands have succeeded as far as securing a contented well-conducted colony is concerned. Even the most successful are not self-supporting, and when the amount of voluntary subscriptions and interest on capital outlay is added show a high cost per head.

In this country conditions for village settlement are much less satisfactory and much less attractive than in England, where every comfort and convenience can be readily obtained in close proximity to large towns whither the villager can escape from time to time for distraction. Here the settlement must be so primitive that it will fail to attract any but the most earnest seekers after health.

A more practical and economical plan would probably be to allow these patients to remain in their homes, to arrange for them to take up part-time work and to continue to pay them an invalid pension, if necessary on a more liberal scale, until such time as it was proved that the disease was permanently arrested. Here, of course, we are confronted with public prejudice and fear, and the despotism of unionism which leaves no place for the slow or maimed worker. The institution in a suitable suburb of workshops and small factories directly under control of the anti-tuberculosis bureau where work could be carried on under medical supervision would meet the case, and would allow the patient's family to pursue their usual avocations, which would be impossible in a remote village settlement.

The careless carrier and the advanced open case also offer a difficult problem. Though against granting power to remove all open cases to institutions, I feel there should be some provision by which the Health Authority could act when notified by a medical practitioner or the dispensary officer that a patient in spite of warning is failing to exercise the care necessary to prevent the spread of infection. The mere possibility of removal would have a salutary effect.

Conclusion.

In concluding this necessarily cursory review let me urge:

The paramount importance of early diagnosis and treatment and the necessity for providing means for such diagnosis.

The need for educating the profession in early diagnosis and treatment.

The need for educating the public in a saner and more accurate knowledge of the degree of infectivity and methods of infection.

The need to remove invidious distinction from the consumptive and the need to provide accommodation for him.

The need for the institution of more dispensaries and sanatoria, more particularly the former, and the need for greater efficiency in their conduct.

The need for careful selection of executive, doctors and nurses.

The need for a scheme, other than village settlements, to utilize the work of the partially fit.

The need for a plan to enable infected but not necessarily affected contacts to get holidays and so nip the disease in the bud.

The need to concentrate the whole effort under a Board with power to control and dictate the policy of the existing individual organization.

The need for power to deal with the careless consumptive.

All this has often been urged before. From repetition sometimes springs conviction and later action.

SOME OBSERVATIONS ON CONTACT WORK IN A TUBERCULOSIS BUREAU.

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It is now over forty years since Sir Robert Phillip first started the idea of a tuberculosis dispensary as the keystone of any anti-tuberculosis campaign, and Edinburgh was the first British University to institute a Chair of Tuberculosis. Sir Robert Phillip is one of the few men who have lived to see his ideas adopted and applied in all civilized countries.

The work of a tuberculosis bureau falls naturally into several subdivisions. These may be briefly summarized as follows, the Bureau becoming: (i) a clearing house for all tuberculous patients, (ii) a centre for diagnosis where patients with doubtful infections may receive proper investigation, (iii) a centre for observation of "contacts," (iv) a centre for certain forms of treatment, (v) a centre for further care of discharged patients, and (vi) a centre for the spread of information to the public concerning the disease.

We have chosen number iii as the subject of our paper, partly because on the proper and systematic supervision and investigation of contacts depends the ultimate success of the tuberculosis dispensary unit in lessening the incidence and mortality of the disease. On looking casually at three or four recent handbooks on tuberculosis, we have been unable to find any mention of the necessity for examination of contacts in dealing with the disease. Moreover, the general practitioner seems to be entirely unaware of the importance of informing the relatives of the desirability of examining contacts in the family when he makes a diagnosis of tuberculosis.

It is, then, more or less obvious that a large proportion of our work must be found in the children, relatives and workmate contacts of patients with pulmonary tuberculosis. Thorough supervision of all contacts both by domiciliary visits and by personal examination is indicated.

Dealing with the contact work of a tuberculosis dispensary, certain considerations arise.

Extent of Work Done in Contact Examination.

The ideal is to examine every contact of every known tuberculous patient, but considerations of limited staff and expense arise. In Melbourne it is our custom to keep a loose-leaved alphabetically indexed sputum book. We confine our activities as a routine to examination of all contacts of persons whose sputum is found to contain tubercle bacilli, the result of examination and reexamination being entered on a card with the following simple headings:

CONTACTS. LIST OF CONTACTS, 19

Infecting Case No.

Address

Name.	Age and Sex.	Date Examined.	Result.	Remarks.

Machinery for Bringing Contacts up for Examination.

On examination of the primary patient at the Bureau, the position is explained, and the need for contact examination urged. The following leaflet is handed to the patient, and serves to fix the examining physician's remarks on the patient's mind.

LEAFLET FOR "CONTACTS."

Keep for Reference.

Most people, at one time or another, have been subjected to infection by the Tubercle Bacillus. Often this leaves no ill effects, but, at times, and in certain people, it leads to actual disease. It is important to recognize the first signs and symptoms of this disease, so that early treatment may be taken. In pulmonary tuberculosis, early treatment is the secret of success. These notes apply particularly to persons who have been in contact with a patient suffering from the disease, or who have had to live in a home where a previous case has existed.

It is suggested, then, that the following signs and symptoms be particularly noted, and that, where two or more of them occur, a visit be paid immediately to the family doctor, or, through him, to the Bureau. Early investigation will almost invariably save future serious disability and trouble.

1. *Irritability of Temper.*—The patient feels badly adjusted to his environment and is peevish, irritable, and sometimes difficult to deal with.

2. *Repeated Coughs and Colds,* which "fly to the chest," and seem difficult to throw off. While these may be simple catarrhs, yet such cases require careful observation lest the tubercle bacillus be stealthily at work.

3. *Pleurisy.*—Probably about 80 per cent. of all definite pleurisies are of tuberculous nature. Too often such patients have a few weeks' rest and return to work, only to break down with flagrant consumption months or even some years later. Such cases of pleurisy should be sent to a sanatorium for at least six months.

4. *Pains in the Chest* may be associated with No. 3, and are probably due in most cases, to small localized attacks of pleurisy. The inflammation and roughening of the lining membrane (Pleura) of the lung and chest cause pain.

5. *Bronchitis.*—Every patient with Bronchitis which does not soon pass off should have his sputum (phlegm) examined lest the tubercle bacillus be present.

6. *Hæmorrhage.*—Bleeding from the lung occurs early in the disease and it occurs late. It is the early type we are concerned with here. Often a small hæmorrhage from the lung is the first and only sign of consumption, and it is a sign of great importance.

7. *Slight Rise of Temperature,* usually in the evening.

8. *Loss of Energy.*—This is an insidious symptom. The patient finds himself tired out in the early afternoon, and requires an effort of will to carry on.

9. *Loss of Weight.*—This may not occur early, but often it does. The patient's friends point this out. In children and young people inadequate gain in weight may be noticed.

10. *Anæmia.*—In every case of anæmia or bloodlessness when the patient does not improve with a course of iron, tuberculosis should be suspected.

11. *Pneumonia.*—In cases where the patient's recovery from pneumonia is slow, tuberculosis must be suspected.

The tubercle bacillus can apparently make headway in a lung damaged by Pneumonia, and often does.

The above are the chief early symptoms and signs of consumption. As the disease progresses, the diagnosis is only too clear, the racking cough, marked loss of weight, sputum, pallor, breathlessness, and night sweats complete the picture.

The great majority of cases of consumption, if taken in time and efficiently treated, get well. Unfortunately, the majority of cases, when they *do* seek advice, are not in the early stages of the disease.

12. Members of the public who live in close contact with a case of lung tubercle should realize the necessity of placing themselves under the doctor for examination and for continued *observation* and *re-examination,* so that if the need arises proper *investigation* of their case may be carried out to check the ailment at the point where *infection* is about to become *disease.*

On notification by the general practitioner, the health inspector or health visitor of the municipality concerned visits the house, and, after instructing the patient in the necessary hygienic measures, leaves the above contact leaflet. A form containing particulars of the house and its inmates is forwarded

to the Bureau, and, when contacts from that house are not forthcoming, a nurse goes from the Bureau and endeavours to make an appointment for examination of the family. If these measures fail, the Tuberculosis Officer visits the house in person, and usually succeeds in examining at least some of the family. For the convenience of workers, an evening session is arranged at the Bureau. Examinations as far as possible are arranged by appointment.

A most important item in the office equipment is the "Memory Tickler." A suspicious contact told to report in three months is registered on a small card which is filed three months ahead. The clerk of the Bureau is reminded automatically to send for this patient, and does so by printed form.

In addition to the above, the Bureau nurses endeavour to revisit every notified patient at least once a quarter, and on these visits she tries to arrange appointments for contacts previously unexamined. It should be clearly understood that this refers to work done amongst the poorer sections of the community. Where a family has a medical attendant, the contacts are told to report to him periodically for examination and reexamination. Cooperation between the general practitioner and the Bureau on these lines, with the gradual education of the public as to the necessity for the supervision of contacts, are the factors which will lead to a definite reduction in the mortality from this disease.

While the original concept of Sir Robert Phillip—the classical "march past" of the contacts—was excellent in theory, in actual practice tuberculosis officers soon found that an enormous amount of time was being expended on "toddlers" and children of school age, while in many anti-tuberculosis schemes failure to arrange for regular evening sessions allowed the most important contact of all—the adolescent—to escape.

Lung tuberculosis is enormously difficult to diagnose in very young children and is impossible in many instances during the hurried examinations if the "march past" policy is strictly adhered to. More satisfactory results, both to the community and the child concerned, may be secured by restricting contact examination of young children to those selected by the nurses as children not thriving. Such children should be carefully observed and thoroughly investigated by the medical officer. As a policy, this frees the hands of the Tuberculosis Officer for the more important young adolescent type of contact, and allows him to seek out the often missed middle-aged or senile "carrier" in the home.

From the administrative point of view, the problem of the contact of school age could very easily and economically be solved by referring all child "suspects" from a house in which a patient with sputum containing tubercle bacilli lives, to open-air classes for a period of three to six months as a routine measure. Such open-air classes should be part of every school and should deal with all children who require special care.

Proportion of Contacts Found Infected.

In the routine work of a Bureau the results of contact work fall under three headings: (i) Cases in which a definite diagnosis of clinical tuberculosis is made. (ii) Cases in which signs and symptoms are suspicious, and in which prolonged observation is indicated. (iii) Cases in which the contacts are apparently healthy, these, as would naturally be supposed, forming the bulk of the cases.

One of us (J. B. F.), between the years of 1919 and 1925 in an English Midland town of approximately 80,000 inhabitants, examined 651 contacts from 433 infecting patients. Eighty-seven were found to be tuberculous, 195 suspicious, and in 369 cases nothing abnormal was detected. This gives a percentage of 11·8% positive infections, with 29·9% suspicious. Details of these examinations are appended (see Table I).

TABLE I.

Year.	Infecting Patients.	Number Contacts Examined.	Positive.	Suspicious.	Normal.
1919	61	75	25	23	27
1920	71	127	22	28	77
1921	51	80	7	50	23
1922	81	85	12	20	53
1923	62	83	11	22	50
1924	49	93	6	24	63
1925	58	108	4	28	76

The statistics in Table II from recent contact and general survey work may be of interest.

TABLE II.

Place and Year.	Infecting Patients.	Number Contacts Examined.	Percentage Positive.	Percentage Suspicious.	Percentage Normal.
Smethwick, 1919–25 ..	433	651	11·8	29·9	58·3
Birmingham, 1925	—	1,970	24·4	—	75·6
York, 1913	75	171	29·8	16·9	53·3
Hull, 1927	—	184	28·1	7·1	64·8
Liverpool, 1926	—	164	10·9	—	89·1
Manchester, 1927	—	667	5·7	—	94·3
Bethnal Green, 1927 ..	—	262	0·8	2·3	96·9
Leeds, 1927	—	616	20·3	24·1	55·6
Massachusetts, 1926 ..	—	1,557	5·29	12·8	81·91
Melbourne, 1927–29 ..	311	741	10·2	16·3	73·5
Norbotten, Sweden. *Isolated community examination.*					
1906	—	1,498	12·75	5·9	81·35
1908	—	1,652	14·0	4·0	82·0
1926 (after anti-tuberculosis measures) ..	—	1,834	8·0	—	—

The following are the figures for Lancashire for the four years 1925–1928, extracted from the Annual Report of the Central Tuberculosis Officer. Averaging for the four years, it was found that 7·2% of contacts were definitely tuberculous, of whom 4·8 were suffering from lung tuberculosis. Detailed particulars of this examination are shown in Table III.

Of the definitely tuberculous contacts over 31% had tubercle bacilli in the sputum.

In 1929 an examination of high school children in Philadelphia showed that at least two in every hundred apparently healthy school children between

TABLE III.

Year.	Number Examined.	Definite Tuberculosis.		
		Pulmonary.	Non-Pulmonary.	Total.
1925	1,221	56	23	79
1926	1,204	46	26	72
1927	898	66	36	102
1928	1,320	54	27	81

the ages of twelve and eighteen years had pulmonary tuberculosis recognizable by symptoms and signs or by X ray examination.

It will be seen that the average incidence of tuberculosis amongst contacts is approximately 12%.

There is ample evidence as to the value of contact work as exemplified in the work of Fröhlich, Asserson, *et cetera*.

Fröhlich's survey of 2,990 children in Oslo was conducted over a period from 1912 to 1927. Of children exposed to tuberculous infection in the family before the age of ten, he found 13·7% subsequently developed tuberculosis. Of those exposed after ten years he found 22·5% similarly suffering. In the survey of 1,520 children not exposed to family infection over a similar period, he found a positive percentage of 10·8% only.

Asserson in 1928 reported on the after-histories of 341 infants. Of 154 contacts, 54% developed clinical tuberculosis and 56% died, the main cause of death being one or other form of tuberculosis. Of 187 non-contacts, 18% only developed clinical tuberculosis and 35% died.

Lemaire in 1925 reported three deaths amongst sixty-seven infants who were separated from a tuberculous family, whilst twenty-one died out of thirty-three, in which the tuberculous environment was maintained.

Ultimate Disposal of Contacts.

The further supervision of contacts apparently normal depends on the amount of staff available, but it is unwise to lose sight of these altogether, and they should be encouraged to report to the Bureau or to their own doctor in case of any unusual or prolonged ill-health. Attention is called to the usual danger signals by the leaflet above. In adolescents it is advisable to continue observation for at least five years.

Examination of Contacts of Patients with Non-Pulmonary Tuberculosis.

In non-pulmonary tuberculosis as a rule one seldom finds positive contacts, with one notable exception, that is, death notification of infants and young children from meningeal and generalized forms of tuberculosis. It is our custom at the Bureau to take special notice of such deaths each month, and to make a particularly careful inquiry into all possible contacts. This is the type of case in association with which one frequently finds the tuberculosis "carrier" usually an adult contact with "asthmatical attacks" or "bronchial catarrh." Care has to be taken not to overlook visiting relatives in such cases—grand-parents, uncles *et cetera*.

A few clinical examples of contact work may be of interest:

Meningitic Type.

A child, aged three years and four months, died from tuberculous meningitis on April 20, 1929. The father came to the Bureau by invitation. He had suffered from asthma since five years of age. The last few years he had been free from asthma, but had had a hæmoptysis in 1926 and dry pleurisy in 1928. He had no other symptoms. Radiological examination at the Bureau revealed extensive bilateral pulmonary tuberculosis.

Pleuritic Type.

A female patient visited the Bureau on September 12, 1928, and found to be suffering from advanced phthisis. The family was investigated, and two sisters, aged seventeen and fifteen years, were found to have had pleurisy with effusion one and two years previously respectively. Both were examined by X rays, and definite signs of apical tuberculosis were detected. Both were given sanatorium treatment, with practically complete arrest.

These two cases probably would not have been detected had domiciliary visitation not been carried out.

A young female patient with sputum containing tubercle bacilli was reported in 1928. At intervals during the next twelve months first a young friend, house and work contact, and then a younger sister, reported, both complaining of chest pains. Both were found to have pleuritic effusions, and were put under observation. During the past few weeks another child, a boy of seven years, has been found to be suffering from pulmonary and abdominal tuberculosis.

Carrier Type.

A female, aged twenty-eight years, was notified as suffering from pulmonary tuberculosis, and visited by a medical officer and nurse in 1929. It was elicited that a brother had died in 1922, and a sister in 1926, both from pulmonary tuberculosis. The mother, aged seventy-three, stated that she had had a cough for many years. She was examined and a specimen of sputum was taken which was returned as positive for tubercle bacilli.

Here the mother was probably suffering from the antecedent infection of the carrier type.

In 1924 a young man died from pulmonary tuberculosis. Three years later his mother reported, and was found to have signs of extensive chronic pulmonary tuberculosis with mild symptoms of many years standing. Her sputum was loaded with tubercle bacilli.

This is a similar type of case to the above.

Familial Type.

The following case is of interest owing to the indefinite nature of the primary infection:

The father of the family died in 1922, probably from pulmonary tuberculosis. In 1926 a relative from the country with sputum containing tubercle bacilli stayed in the house and slept in the same bed as two of the daughters. In 1927 one of these daughters was admitted to sanatorium, and died shortly afterwards. In 1928 two other daughters, aged twenty-two and twenty-five, were examined. The sputum of both contained tubercle bacilli. A visit was paid to this family in August, 1928, and the following was found: The mother had a suspicious right apex. A daughter, aged twenty-five, had a definite infection. A daughter, aged twenty-four, had a definite infection. A daughter, aged twenty-two, had a definite infection. A daughter, aged twenty-one, was away from home. A son, aged sixteen, had a suspicious right apex. A son, aged fourteen, was apparently normal. A daughter, aged eleven, had a suspicious left lung; later the condition rapidly advanced. A daughter, aged ten, was poorly developed and mentally defective.

It is difficult to state definitely at what stage infection was introduced, but the need for family observation is apparent. This family came from

country to town a few years ago, and illustrates familial lack of resistance in country-bred people exposed to town conditions. An interesting "side-line" in this case is that the *fiancé* of one of the daughters was also recently reported to have had an attack of "blood-spitting." This family persistently failed to cooperate with the Bureau.

Early Diagnosis.

This patient (number 143) has an advanced open infection: he was nursed by his mother. It was suggested on a domiciliary visit that the mother should be examined at the Bureau. She reported slight cough and huskiness for two months. She was examined by X rays and her sputum was tested; both investigations revealed definite pulmonary tuberculosis.

Without a system of domiciliary visiting, this patient's condition would probably have been far more advanced before she consulted a medical man.

A female patient with an open infection, when paid a domiciliary visit, stated that her brother had had a cough for several months. The young man himself was found doing physical exercises in the back yard and was persuaded to come to the Tuberculosis Bureau for overhaul. Sputum was found to be positive for tubercle bacilli. Beyond his cough, he showed no symptoms, and would probably have neglected seeking medical attention until symptoms of the disease appeared, had he not been asked to attend the Bureau. This young man had a course of sanatorium treatment and is at present doing very well.

Familial Lack of Resistance with a Carrier.

Three fatal cases of pulmonary tuberculosis occurred in a patient's house. The patients were visited by the medical officer. The father, a man of seventy-two, stated he had had a winter cough for seven years. On examination, his sputum was found to be loaded with tubercle bacilli. The one daughter remaining was examined by X rays and found to show signs of apical disease.

The father was probably the infecting patient, and under a scheme of examination of contacts, would probably have been detected when the first fatal case occurred in 1924. If this had been done, the three later patients might have been saved. The whole family is now dead.

Illustrating Neglect of Advice.

A patient was visited early in 1926. The father was found to be suffering from advanced pulmonary tuberculosis with positive sputum. There was a family of seven children. The eldest girl gave a history very suggestive of early pulmonary tuberculosis. She was irritable, tired and generally run down. Her mother was advised to bring her to the Bureau, but this was not done. Two years later this girl was reported as a definite sufferer from pulmonary tuberculosis, and died in a further eight months.

It is felt that, owing to the smallness of staff at that time, insufficient pressure was brought to bear in this case.

Criteria of Diagnosis.

It is to be borne in mind that we have to differentiate between infection and disease. To understand the disease tuberculosis, as opposed to infection, it is well to remind ourselves of Koch's phenomenon that skin and subcutaneous tissue of an animal infected for the second time with tubercle bacilli react in a way which is quite different from that following the first infection. A primary subcutaneous dose of tubercle bacilli into an animal produces no changes and no symptoms until two weeks later, when the nearest lymphatic gland enlarges. If some three weeks after the first dose a second dose is given, the skin at the site of the inoculation becomes inflamed within twenty-four to thirty hours, and a hard nodule forms underneath, accompanied by severe clinical signs. A week later sloughing occurs. The mechanism of this phenomenon explains the principles involved in human lung tuberculosis, both as regards the lesion and the symptoms produced. In other words, the primary infection has sensitized the patient. He becomes allergic after the primary tubercle has undergone degenerative changes which liberate diffusible products into the circulation. The tuberculosis physician must realize that in almost every case of lung tuberculosis there was a time at which ordinary infection was being repeated or augmented in such a way that the condition was becoming one of clinical disease. He must concentrate his attention, particularly as far as contacts are concerned, on the stage when slight deviations from the normal, many of them of a reflex nature, indicate that some such allergic process is at work. Symptoms are of more importance than signs in early diagnosis, and it is useless in these cases to wait until marked signs are present.

The symptoms which are most valuable have been outlined in the contact leaflet above, and need not be repeated here. Clinical history must be thoroughly and conscientiously taken. A complete anamnesis is the first and most important step.

In considering signs the following facts should be remembered:

1. Light percussion is most valuable.

2. Krönig's isthmus, when carefully elicited, is of value, if the possibility of error from catarrhal atelectasis is borne in mind.

3. Sputum examination and examination of fæces should be conscientiously carried out. In young children if the mother is armed with a throat swab, she may obtain a positive specimen by swabbing the pharynx after a spasm of coughing. It is well known that children tend to swallow their sputum.

4. Harsh breath sounds just external to the nipple region and in the axillæ of children are often suggestive of spread of disease from a hilum.

5. The egg-shaped area of paravertebral dulness between the third and fifth dorsal vertebral spines is asymmetrical and enlarged to one or other side in hilus disease. Careful percussion and practice will elicit this most valuable sign (Ewart's area). D'Espines's sign and the Eustace Smith murmur have not been found of direct diagnostic import. In eliciting whispered pectoriloquy, we have found the word "Leicester" useful.

6. In adolescents, dilated venules on either side of the upper dorsal spine (the *zone d'alarme* of the French) are of considerable value in arousing suspicion of clinical tuberculosis. Enlarged venules, most noticeably in the second interspace, particularly when limited to one side of the chest, mentioned by Still, may also help considerably in diagnosis.

7. Increased muscle tension often slight in degree may be found particularly in the trapezei, rhomboid and pectoral muscles. This sign often persists between exacerbations of symptoms. A certain amount of practice in light palpation is required in

detecting the sign, and we should like to emphasize its value.

8. There are often no demonstrable physical signs, in which case radiography may or may not help. It is somewhat disconcerting when, for some reason or other, contacts have had stereoscopic radiographic examinations made with no visible abnormality in the films, to find that in several instances pleurisy with effusion has developed within a few weeks, or other definite signs have declared themselves. After all, this is not to be wondered at if we remember the pathology of the disease, and the primary subpleural focus put forward by Ghon and Kuss, Opie, Petroff and others.

In short, a diagnosis can be arrived at only by prolonged observation and investigation, reviewing all the factors, family history, exposure to infection at home or at work, symptoms, signs and careful X ray examinations. If doubt still remains, it is our custom to subject the suspect to a series of diagnostic tuberculin injections, with either old tuberculin (T.) or albumose free tuberculin (T.A.F.).

Our usual dosage in children is 0·0001, 0·0005, 0·001, 0·0025 and 0·005 cubic centimetres. In adults, the series is 0·0005, 0·001, 0·0025, 0·005, 0·0075 and 0·01 cubic centimetres.

Our opinion is that: (i) When tuberculin is properly employed with due skill and care, no harmful results arise. (ii) In the absence of a tuberculous focus within the body, no tuberculin reaction can occur with the dosage used.

Essentials of the Test.

1. The test should be used only in cases in which the temperature curve does not exceed 37·2° C. (99° F.) in the evening (five minutes' mouth temperature) and the chart should be kept for one week before and during the making of the test.

2. The test should not be used when signs and symptoms are indicative of tuberculosis, and when the test is superfluous.

3. Its use should be avoided when there has been recent hæmoptysis.

4. Its use should also be avoided in myocardial disease, or in persons with damaged hearts with poor compensation.

5. Renal disease, hysteria and epilepsy are also contraindications.

Tuberculous Diathesis.

It still remains a puzzle why certain infected individuals develop clinical manifestations, whilst in others the disease remains latent. Factors at work may involve the nervous system, organs of internal secretion, circulatory system, and digestive organs, all of which have a profound influence on the general metabolism of the individual. There is no doubt that the rate of metabolic activity varies in individuals, and this must bring into play many factors which influence resistance. Discussing these points under the various systems, we should like to call attention to the following frequently found disturbances.

Digestive System.—Whilst dyspepsia is a common complication of lung tuberculosis, it is true conversely that dyspepsia may so lower resistance that it may determine lung tuberculosis. Apperly has shown that the secretion of hydrochloric acid is on the whole much less in the consumptive than in the normal person. Our knowledge about the pancreas is less definite, but nutrition may be deficient as regards fat digestion. The tuberculous "cannot eat fat," and tuberculosis is a common complication in diabetes. The recent work of Minot and Murphy on liver feeding in pernicious anæmia seems to suggest that the liver possesses some substance which deals with the well-being of the blood itself. How seldom do we see extensive tuberculosis in the human liver at autopsies when tubercles are widespread elsewhere.

Respiratory System.—Marked differences are found in individuals in the mode of breathing, the elasticity of the lung itself, and in the power of the lung to form fibrous tissue in the face of infection. These factors must play a large part in deciding whether infection shall remain latent or not.

Circulatory System.—How often do we see a small narrow heart in the X ray film of the tuberculous subject? Whether this means a less efficient pump or is associated with a narrow sloping chest is open to discussion. Broadly speaking, the blood pressure, both systolic and diastolic, tends to be lowered in declared tuberculosis. These factors may have been present for some time. A factor of more importance is where the vasomotor control is unstable, with a tendency to bear strain and fatigue badly, and to cause these persons to seek shelter from colds and draughts and shut themselves up in stuffy rooms.

Glands of Internal Secretion.—The glands of internal secretion, notably the thyreoid and adrenals, have a profound influence on metabolism, and the thyreoid especially in resistance to infection. Possibly the tendency to tuberculosis met with in convalescents after measles and whooping cough is associated with thyreoid exhaustion. After influenza the same factor may be at work, *plus* adrenal exhaustion. One of the commonest of the minor signs in early tuberculosis is slight enlargement of the thyreoid gland, particularly in adolescent female patients, often associated with disturbances of vasomotor control.

Nervous System.—Individuals vary in the toughness of their mental fibre, and react differently to the calls of every day. It is quite evident that many individuals suffer from nervous instability, and often the most vivacious and energetic are also most liable to fatigue and nervous exhaustion.

From the viewpoint of organic neurology, an unstable nervous system or one that is easily fatigued must predispose to under-function of many organs and many glands of internal secretion, and interfere with many of the factors involved in the defences of the body. Commonly these defects show as functional disorders of many kinds, with neurasthenic symptoms.

From the psychological point of view, probably much might be learned, not only of the factors which determine the incidence of the disease, but also of those which influence the patient's ability to resist the disease. Where energy is locked up in holding down repressions and complexes of various kinds,

to speak in terms of the "new psychology," one might very well expect neurasthenic symptoms. It is well known that the tuberculous patient is "*difficile*." Broadly speaking, tuberculosis tends to pick out the emotional and imaginative individual. Tuberculosis in the past has been studied largely from the point of view of the infection, of late from the point of view of defence mechanisms, biological and biochemical, but little or no work has been done from the psychological aspect.

In conclusion, we may claim that the great need of the moment in connexion with this disease of lung tuberculosis is the education of the public in the importance of examination, investigation and observation of all contacts by the family medical man.

Reviews.

RADIOGRAPHY OF THE CHEST.

"THE CHEST" is a comprehensive study of disease of the chest, considered from a radiological standpoint, written by L. R. Sante, of St. Louis.[1]

The work is divided into three parts. Part I deals with certain essential preliminaries to chest interpretation, Part II with evidences of pathology as revealed radiographically, and Part III is devoted to a detailed description of each disease from its inception to its termination. Fluoroscopy is recommended as an adjunct to radiography; the former is of value in observing movements of organs and for examination in varied positions, while radiography gives greater detail of information and forms a permanent record. Fluoroscopy should not be employed as a routine without supplementary radiography. Standard postero-anterior, antero-posterior, oblique and lateral views are used for the various studies. Stereoscopic films are of great help. In regard to lipiodol (the technique of administration is described in detail), the supraglottic route is recommended in preference to the injection through the crico-thyreoid membrane, except for children. Chapters are devoted to the anatomy of the chest and to the physiology of respiration. As a rule it is possible to give a diagnosis from the film alone, but in unusual and obscure cases the history is of value and should be correlated with the X ray findings. Asymmetrical chests frequently occur, but should be easily recognizable. The author calls attention to the necessity of fluoroscopy in the examination of the mediastinal contents with the patient in various stages of rotation. It is frequently difficult to distinguish aneurysm from tumour, and the continuity of the abnormal shadow should be established and expansile pulsation demonstrated. The author refers to the difficulty of differentiation between expansile and transmitted pulsation in any tumour. Pathological change in the œsophagus is generally easy to differentiate from other mediastinal conditions. Dunham's "three zones" are used in descriptions of lung conditions: the inner zone occupied by the hilum, the middle zones by the larger bronchi and the other zones by the small feathery markings of the bronchioles and alveoli. The various pathological conditions occurring in these zones are illustrated by clinical drawings of unusual clearness. The author points out that no disease produces hyperactivity of the diaphragm muscle. Various conditions causing alterations in mobility and outline are tabulated and described. Part III deals with various diseases in detail, commencing with the trachea and bronchi and traversing the bronchi, lung tissue, mediastinum and œsophagus. An acute bronchitis causes increased markings along the bronchial tree, due to the presence of secretion in the bronchi, while the markings in chronic bronchitis are due to actual peribronchial thickening and enlargement of hilar glands. Primary bronchial tumours cause bronchial blockage and atelectasis.

Bronchiectasis is lucidly explained, and though it usually follows an acute infection, there is some congenital lack of elastic structure in the bronchial walls. The author draws attention to the extreme rarity of bronchiectasis in the upper lobes. It is easy to miss a bronchiectasis in the lower lobe when situated behind the heart shadow or behind the diaphragm; lipiodol is of great value in these situations. Differentiation from lung abscess is often impossible. The illustrations in the section dealing with lobar pneumonia and bronchopneumonia show the various stages and types. In defining lung abscess, the author includes only acute destruction of lung tissue within the lung itself and excludes tuberculous cavities, empyemata *et cetera*. The Hirtz compass is recommended for accurate localization of lung abscess, this being very essential when operation is to be undertaken. When lung abscess becomes infected by putrefactive organisms, it really becomes a gangrene. The author calls attention to the formation of a pulmonary fibrosis after deep X ray therapy.

An interesting chapter is included on the various modes of dissemination of pulmonary tuberculosis in children and in adults. It is noteworthy that in patients with general miliary tuberculosis there is little if any indication of a preexisting pulmonary tuberculosis. The section on silicosis is well up to date and the fact that silica in the dust is the only causative factor is stressed; coal and other substances do not cause silicosis and any dust is dangerous in direct proportion to its silica content. Röntgen examination is the only method of diagnosing a silicosis. Primary and secondary carcinoma receive consideration in full. The work ends with a description of disease affecting the mediastinum (including œsophageal disease). This work is the most complete seen to date and though of greater value to radiologists, it should be in the hands of every physician who recognizes the value of X ray work in chest disease.

[1] "Annals of Roentgenology: A Series of Monographic Atlases," Edited by James T. Case, M.D.; Volume XI: The Chest, by L. R. Sante, M.D., F.A.C.P.; 1930. New York: Paul B. Hoeber, Inc. Demy 4to., pp. 588, with three hundred and seventy-six Röntgen-ray studies and one hundred and sixty-three clinical illustrations. Price: $20.00 net.

A BOOK ON TUBERCULOSIS FOR NURSES.

IN "A Manual of Tuberculosis for Nurses," a simple yet adequate text book has been provided by Dr. E. Ashworth Underwood for those nursing patients with this disease.[1]

Written as it is for the guidance of nurses, the language is clear and the context should be easily understood, even when, as rarely happens, more difficult aspects are introduced.

The early chapters give an explanation of the principles underlying infection and the reactions caused by such infection. Too much importance has been laid on the risk of bovine infection, while the greater risk of human infection is not stressed. The author tends to the opinion of primary infection in childhood and draws little attention to infection in adult life.

The symptoms and signs of the disease are described simply and in detail, as are its complications and different forms met with. Little exception can be taken as to the advice given for the treatment of each condition.

Considerable space is occupied in pointing out the routine duties of a nurse in a sanatorium, in the home and in district work. Several of the older methods of special treatment are mentioned, also the more recent methods likely to be encountered in practice.

[1] "A Manual of Tuberculosis for Nurses," by E. Ashworth Underwood, M.A., B.Sc., M.B., Ch.B., D.P.H., with an introduction by J. R. Currie, M.A., M.D., M.R.C.P., D.P.H.; 1931. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 279, with illustrations. Price: 6s. 6d. net.

From a long and intimate association with the treatment of tuberculosis in its different phases, the author has appreciated the many difficulties a tuberculosis nurse is likely to meet. His book is an attempt to supply an explanation for and information regarding every circumstance that a tuberculosis nurse may experience in her work, avoiding all controversial and doubtful matter, but keeping strictly to the more simple yet reliable methods already proved satisfactory.

The book should prove of great value to all nurses.

FORENSIC MEDICINE.

DR. RALPH W. WEBSTER, Professor of Medical Jurisprudence in the Rush Medical College, University of Chicago, already has his name attached to one of the best known American text books in forensic medicine, namely, "Legal Medicine and Toxicology," by Peterson, Haines and Webster. He has now written a work with the same title and states that his object is to present in one volume the more usual phases of legal medicine in a somewhat concise manner for the busy practitioner.[1] In this he is successful, except as regards toxicology, to which he devotes much more than half the book—far too much for the busy practitioner. Much of this large amount of space is devoted to the detection and separation out of the poisons, work which, wherever possible, should be left to the expert analyst.

The book being an American work, the legal procedure does not entirely coincide with that of this country. But this is a minor matter and provides points of interest, such as in regard to the question of medical privilege. In New York and some of the other states "a person duly authorized to practise physic or surgery shall not be allowed to disclose any information which he acquired in attending on a patient in a professional capacity and which was necessary to enable him to act in that capacity." Moreover, it has been held that reports from hospital records are privileged in the same way and cannot be introduced in evidence.

Violation by a medical man of the law dealing with the sale and dispensing of preparations of opium and cocaine has been held to constitute gross malpractice.

Discussing paternity, the author shows himself to be a believer in telegony. He even suggests that the question may be confused by the influence of maternal impressions.

Extensive use has been made by the author of many of the best known works on forensic medicine, and on the whole this book can be taken to be a good and up-to-date exposition of the subject.

SURGICAL PROCEDURES.

THE fact that Pye's "Surgical Handicraft" has reached its tenth edition is a sufficient indication of the esteem in which it has been held by successive generations of the medical profession over a period of almost fifty years.[2] This edition is all the more notable since it is the last contribution to surgical literature of so distinguished a surgeon as the late Mr. Herbert Carson. Under his guidance the entire volume has been revised and much new material has been added.

The publishers claim that much obsolescent material in both text and illustrations has been deleted. However, the reader is unpleasantly surprised to find, both in the text and among the illustrations, descriptions of procedures and apparatus which are actually obsolete. This is particularly noticeable in the section on splints. Truly tradition dies hard in these veterans of medical literature.

The new section dealing with infections of the hand is a brief, perhaps too brief, description of methods of diagnosis and procedure in the various types of infection. As the recognition and correct treatment of such conditions are among the common daily tasks of the casualty surgeon, and because incorrect diagnosis and procedure may bring such dire functional results, we should like to see more space devoted to this subject.

Other new sections of note are those dealing with the tannic acid treatment of burns and the injection treatment of varicose veins. The outstanding success of the former and the almost universal use of the latter render these additions of considerable value.

We are not favourably impressed by the illustration depicting the injection of varicose veins. In this aseptic age an illustration of a surgeon performing a supposedly clean surgical procedure with shirt sleeves down, wearing a ring, and resting his hand on the doubtfully sterile stocking of the patient, seems singularly ill-chosen.

The section on venereal diseases has been rewritten and enlarged, and covers this field in a comprehensive manner.

A very short chapter on the radium treatment of cancer gives the bare outline of this method, but, wisely in a book of this kind, omits those details which are the concern of the specialist in this work.

There are two notable omissions. No reference is made to the control of intracranial pressure by intravenous and rectal injections of salines and none to Böhler's method of local anæsthesia in the treatment of fractures. Both of these are in common use and should come within the scope of this book.

The book is well printed on good paper and the illustrations and plates are well reproduced. The index is satisfactory.

As it has been in the past, so in the future will this work be valuable as a guide to the student, house surgeon and practitioner.

ORGANIC COMPOUNDS AND THEIR SYMBOLS.

ANY attempt at simplifying and systematizing chemical notation is worthy of attention, and for that reason "Structure Symbols of Organic Compounds," by Professor Hackh, holds much of interest.[1] After an introduction covering a statement of the why and wherefore of existing methods of formulating organic compounds, the author proceeds to elaborate his own system. The symbols he employs he regards as summarizing in a characteristic and rapidly written form more information regarding the compounds they represent than is available from any other type of symbol at present in common use. The hope is expressed in the preface to the book that the new system will be of help to students and teachers of organic chemistry, and it is stated, moreover, that experience has shown that the system can be learned by a small expenditure of concentrated effort.

It is to be feared that few students or teachers will be prepared to add even so lightly to their burdens by adopting this new notational system. Many of the symbols employed show little or no superiority over those at present in use, especially in the case of those representing compounds containing one or more benzene rings. Again, the limitation of the system to only the four elements, carbon, hydrogen, nitrogen and oxygen, necessitates reversion in other instances to the more common methods of formulation. The new, therefore, cannot replace the old system, but can be used only as auxiliary to it.

The book is interesting as a contribution to the subject of chemical notation, but can hardly be regarded as helpful in the way intended.

[1] "Legal Medicine and Toxicology," by Ralph W. Webster, M.D., Ph.D.; 1930. Philadelphia and London: W. B. Saunders Company; Melbourne and Christchurch: James Little. Royal 8vo., pp. 862, with illustrations. Price: 50s. net.

[2] "Pye's Surgical Handicraft": Tenth Edition; Edited by H. W. Carson, F.R.C.S.; 1931. Bristol: John Wright and Sons. Demy 8vo., pp. 659, with illustrations. Price: 21s. net.

[1] "Structure Symbols of Organic Compounds, an Adjunct to Text Books of Organic Chemistry for Students and Teachers," by I. W. D. Hackh; 1931. Philadelphia: P. Blakiston's Son and Company. Demy 8vo., pp. 147.

The Medical Journal of Australia

SATURDAY, AUGUST 8, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

PULMONARY TUBERCULOSIS AND THE PUBLIC HEALTH.

The causative organism of tuberculosis is known and can easily be recognized. The pathological process consequent on its entrance into the human body and its involvement of the pulmonary tissue is understood. The problem of early diagnosis, though difficult, is not insoluble. The manner in which an infected person spreads the infection among the non-infected is realized by all medical practitioners. The principles underlying treatment are appreciated and many and diverse methods have their ardent advocates. It might appear that no useful purpose could be served by detailed discussion of a subject so often discussed before and about which little that is new can be said. Unfortunately, familiarity, if it does not always breed contempt, often begets indifference and sometimes tolerance. To be indifferent to the ravages of pulmonary tuberculosis is blameworthy and a sign of sloth, to be tolerant of them is criminal. No excuse need therefore be offered for devoting the greater part of this issue of The Medical Journal of Australia to the subject of pulmonary tuberculosis in its relation to the public health. The four original articles have been contributed by special request, and this opportunity is taken of thanking the authors for their valuable contributions.

Dr. J. H. L. Cumpston has shown the extent of the prevalence of pulmonary tuberculosis in Australia. He points out that for purposes of control pulmonary tuberculosis must be regarded as human in origin. His statement that in every thousand people some six or seven hundred are invaded by the tubercle bacillus, that of these some two hundred show visible lesions after death, that fifteen become clinically identifiable as infected, and that 0·5% die of pulmonary tuberculosis, should give pause to the indifferent and the tolerant in a community so richly endowed as Australia, a country which so eminent an authority as Dr. Victor Heiser has declared should be free from tuberculosis. Dr. Cumpston naturally links up pulmonary tuberculosis with other infectious diseases and then raises the questions of notification and the examination of contacts. As far as notification is concerned, Dr. F. S. Hone has shown that the present system is not all that might be desired. We would direct attention especially to his remarks on the notification of non-infectious cases and the provision of adequate control after notification. Dr. Sinclair Gillies's paper follows naturally on that of Dr. Hone. As Dr. Gillies points out, most of what he writes has been written before. His method of presenting his facts, however, should make medical practitioners ask themselves whether they are doing the best for their patients and health departments, whether their administration is sufficiently complete, and whether it calls forth confidence in the minds of medical practitioners. His remarks on the education of the public in regard to the infectivity of the consumptive might well be broadcast. The same statement holds in regard to much that appears in the article of Dr. J. Bell Ferguson and Dr. James. Dr. Ferguson was asked to write this article because the treatment of contacts is probably more neglected by medical practitioners than any other aspect of the treatment of pulmonary tuberculosis. The method described by him has certainly been productive of good results.

Apart from the need for an awakening of general interest in the subject, three points call for special

emphasis. These are the relationship of the medical practitioner to the health authority, the relationship of the health authority to the medical practitioner, and notification. In regard to the first of these, the medical practitioner must realize that he cannot diagnose pulmonary tuberculosis and treat the patient without any reference to the health authority. It may be asked in all seriousness whether the average medical practitioner is qualified to diagnose and treat every case of pulmonary tuberculosis. If he is wide awake and knows his medicine, he will probably be equal to the requirements in most instances. Sometimes the best informed general practitioner needs the assistance of a specialist, and even a learned specialist may need the help of another. It is here that the antituberculosis dispensary comes into the picture. Dr. Gillies has given a good account of the place which the antituberculosis dispensary should occupy. Through this agency the practitioner comes into relationship with the health department. Sometimes the relationship may be direct. The medical practitioner must realize that neither the antituberculosis dispensary nor the health department officer wishes to take his patient from him. He must also know that occasions will arise when the patient must be passed to someone else's care. In this matter the welfare of the patient must be the only consideration. What the health department officers desire is that their resources shall be used with those of the medical practitioner. They wish to work with him and not against him. He must provide the opportunity. Thus arises the second point, also mentioned by Dr. Gillies. If the aid of the health department is to be sought by the medical practitioner whenever it appears to him necessary, the health department must have at the head of its antituberculosis activities a man who knows every aspect of his subject and who can command the respect of every private medical practitioner. In regard to the last point, notification, the subject must be reviewed in the light of such views as those expressed by Dr. Hone. The Federal Health Council discussed this matter in 1929. It made certain recommendations along the lines suggested by Dr. M. J. Holmes and referred to by Dr. Hone in his paper. The recommendations have not borne fruit. Possibly something definite might be done, if the whole question were referred to a special conference of health authorities and practising members of the medical profession. Recently conferences have been held on cancer treatment and poliomyelitis and a better understanding of the problems presented by these subjects and of the steps necessary to their solution has resulted. At such a conference an extension of the recommendations of Dr. Holmes might be considered. Thus additional information might be required on the notification form, possibly concerning the type of infection, the recommendations of the medical attendant as to home, hospital or sanatorium treatment, and in regard to contacts. A somewhat similar suggestion was recently made in these columns in reference to scarlet fever. The idea is not that medical practitioners shall be dragooned into following a certain set course of action, but that the information provided shall be a starting point in the necessary cooperation between health department and private medical practitioner. It would be useless to contemplate such extensions of the present system if the medical profession was not prepared to cooperate and if the health department, both as far as personnel and organized activities were concerned, had not the entire confidence of the practising members of the medical profession.

Current Comment.

THE INTRACUTANEOUS TUBERCULIN TEST.

Tuberculosis has been one of mankind's deadliest enemies since the beginning of history. Thanks to modern methods of hygiene and sanitation, it is not the lethal scourge, nor does it commonly present the loathsome manifestations with which our forefathers were familiar, but it is still ubiquitous and, while it most readily claims as its victims people who live in unhealthy surroundings, it may attack people anywhere and at any time. This fearsome disease, which influences the lives of so many people, controls the lives of so many more and kills such vast numbers so indiscriminately, has naturally been subjected to an enormous study, both in the matters of diagnosis and treatment. Formerly diagnosis of tuberculosis was often only possible when the disease was so far advanced that there was little prospect of recovery; on the

other hand, people were often wrongly stigmatized as tuberculous. Koch's discovery of the tubercle bacillus led eventually to the introduction of various tuberculin tests, which caused further confusion and the chronic invalidism of many unfortunate people who were actually quite free of active tuberculous disease. It was realized at last that such a test as the von Pirquet test gave information only as to whether any portion of the body had ever been invaded by the tubercle bacillus, and not as to whether there was any active disease at the time of the application of the test. It was of value only when no reaction was elicited. Thus tuberculin tests have been wholeheartedly condemned as useless and misleading by some authorities, but there are many who believe that these tests, or some of them, have a value in the diagnosis of tuberculous disease. The percutaneous test and the tests of Calmette and von Pirquet, the subcutaneous test and the elaborate test of Ellermann and Erlandsen *et cetera*, all have their advocates. No doubt each has some value in its proper sphere. The intracutaneous test, introduced by Mendel, has come to be associated with the names of Mantoux and Roux; as with all the other tests, there is considerable divergence of opinion concerning its value. The clinical application of this so-called Mantoux test is the subject of a recent communication by Alan Moncrieff of the Hospital for Sick Children, Great Ormond Street, London.[1]

Moncrieff complains that while previous investigations have led to considerable increase in scientific knowledge of the intradermal test and its interpretation, there has been but little attempt at analysing its value to the clinician faced with some problem of diagnosis.

He remarks that the ideal test for tuberculosis should be easy to perform accurately; it should be free of all harm to the patient and the results should be easy to read and to interpret. While no test fulfils all these requirements, he affirms that the Mantoux test most nearly approaches the ideal, save in the matter of interpretation. It has obvious advantages over the von Pirquet test, not the least of these being that it allows of the administration of tuberculin in an accurate dosage. It is estimated that about 0·01 milligramme of old tuberculin is given by means of the von Pirquet method; in the application of the Mantoux test, ten times this amount is administered as an initial dose, thus, according to some workers, some persons react to the Mantoux test who would not react to the smaller dose employed in the von Pirquet test. It may be pointed out here that this is not in accordance with the views of some others, who declare that the degree of severity of a local reaction depends on the concentration of the tuberculin suspension rather than the quantity.

For the purpose of his investigations Moncrieff submitted to the Mantoux test 206 patients in the Hospital for Sick Children and correlated his findings with the final diagnosis. He admits that this diagnosis may have been occasionally wrong and that apparently non-tuberculous children who reacted to the test may ultimately be found to be tuberculous. He remarks, however, that this possibility is of no significance in his investigation, as his sole concern is an estimation of the value of the Mantoux test as an aid to diagnosis. His line of reasoning here is rather difficult to follow. Surely disregard of a positive reaction and a consequently incorrect diagnosis are indicative of a false estimation of the value of the test. Eighty-eight patients in whom there was no apparent evidence of tuberculosis were selected as controls; in the remaining 118 instances the test was employed for the purpose of diagnosis. The youngest of the control children was four weeks old and the eldest twelve years. Twelve of them reacted to the intradermal injection of tuberculin. None of the controls who reacted was younger than seven years. The administration of several increasing doses did not evoke a response from six of the patients. Moncrieff regards this as evidence that the use of repeated small doses of tuberculin does not result in sensitization as has been suggested by some workers; Calmette has stated that sensitization may be effected in this manner, but only in persons who have been already infected with tuberculosis.

Of the twelve control children who reacted to the test, eight reacted also to the control injection of glycerin-bouillon. There are numerous possible causes for this; it has even been suggested that such reactions may be due to the use of broth made from the flesh of tuberculous animals. At any rate, it is clear that a control injection should always be employed. In the remaining four instances no control injection was used. Moncrieff remarks that in this series reaction to the control injection was a rarity.

Moncrieff divided his patients into eight groups according to their various illnesses: patients suffering from "chest conditions," "abdominal conditions," "intracranial conditions," "febrile conditions," "enlarged glands," "kidney affections," "bone affections" and "miscellaneous" conditions.

Of forty-four patients suffering from some affection of the lungs or air passages, seven were afflicted with pulmonary tuberculosis; six of these reacted to the Mantoux test; the seventh was very ill and did not react. Fourteen others in this group also reacted. In several instances a reaction was deceptive; if reliance had been placed on the test, two young children affected with bronchitis would have been regarded as tuberculous.

As two children who were undoubtedly affected with abdominal tuberculosis did not react to the test, the question arises as to whether bovine tuberculin should not be employed in such cases. It is of interest to note also that two children suffering from spinal caries did not react. On the other hand, two children suffering from cerebral tumour and one affected with lymphadenoma reacted. One other patient who reacted to the test died; a *post mortem* examination revealed no evidence of tuberculosis.

[1] *The Quarterly Journal of Medicine*, January, 1931.

Moncrieff concludes that there are few instances in which the Mantoux test is of value. The value of the absence of any reaction is reduced by the possible existence of several agencies tending to influence the result of the test. For example, the use of bovine tuberculin may be necessary to evoke a reaction; an acute illness, whether tuberculous or not, may prevent a reaction, or the tuberculin may be impotent. Moncrieff points out that a reaction means no more than that the individual on whom the test is applied has at one time or another been infected by the tubercle bacillus, but he remarks that doubt is thrown on even this conclusion by the absence of any evidence of tuberculosis in one of his patients examined *post mortem*. He advises restraint in the application of the Mantoux test for diagnostic purposes, and states that a reaction even in very young children should not be accepted as proof of active tuberculosis, but should be considered merely in conjunction with other evidence and be disregarded if necessary.

Of course, failure to find evidence of tuberculosis at a *post mortem* examination does not by any means prove that no infection has been introduced; by a study of serial microscopical sections of the whole body, it might be possible to exclude tuberculous infection, but even this is doubtful. On the other hand, it is not inconceivable that a person may become sensitized to the toxins of tubercle bacilli without suffering from an actual infection. Thus some reactions may be accounted for. It is possible that the failure of some tuberculous persons to react may be explained according to Moncrieff's views.

Most authorities agree that absence of a reaction signifies absence of infection save in instances of severe illness, and that a reaction in infancy is strong evidence of the existence of actual disease. Probably Moncrieff's view is more nearly correct in regard to the latter.

Medical practitioners should have a knowledge of all the commoner tests employed in the diagnosis of tuberculosis. Different tests should be used in different circumstances; when there is evidence of tuberculosis and confirmatory evidence is required, it may not be advisable to employ the same test as when it is desired to exclude tuberculosis. For example, it is believed that very few healthy people react to the conjunctival test, and that some people actually suffering from tuberculosis do not react; thus a reaction to this test may be regarded as of greater significance than a reaction to von Pirquet's test. Of course, the most important aid to the diagnosis of pulmonary tuberculosis is radiography, but this is not readily available to all people.

Moncrieff's paper contains so much that is worthy of consideration and presents such a vast number of interesting possibilities and problems that it is manifestly impossible to discuss it fully here. The paper should be read by all who have the opportunity; it should be read critically and should not be accepted as the last word on the use of tuberculin in diagnosis.

TUBERCLE BACILLI IN THE GASTRIC CONTENTS.

THE diagnosis of early pulmonary tuberculosis in infants and very young children often presents very considerable difficulties. Though in the examination of children radiography is of special diagnostic value, there is a commencing period in most, if not all, instances of tuberculous infection during which no opacities are revealed by the X rays. Such a condition is of course due to the absence of fibrous tissue; perhaps at this stage the tubercle bacilli have so much the better of their combat with the body defences that there is no time for the formation of fibrous tissue. Often the careful and discerning physician may be able to find clinical evidence sufficient to justify making a diagnosis of pulmonary tuberculosis, but such evidence may be absent. In either event there is an absence of certain diagnostic evidence. In such instances the examination of the fæces for the presence of tubercle bacilli has been advised, for infants and very young children swallow their sputum. But the existence of a tuberculous enteritis may interfere with the correct interpretation of the results of such an examination. It is interesting, therefore, to read a recent paper in which Valdemar Poulsen discusses the diagnosis of pulmonary tuberculosis in children by means of bacteriological examination of the gastric contents.[1] He remarks that in many instances of pulmonary tuberculosis of children physical signs cannot be elicited and the radiological examination reveals either no abnormality or a condition varying so slightly from the normal that a diagnosis cannot be made unless tubercle bacilli be found in the sputum. In such instances he employs gastric lavage, examines the sediment in the washings and, if necessary, inoculates Petroff's medium or injects a portion of the material into a guinea-pig. The possibility of the existence of tuberculous disease of the stomach is remote; a tuberculous gland at the tracheal bifurcation sometimes becomes adherent and empties its contents into the œsophagus. Both these contingencies may be disregarded as a general rule. If, then, the child's tonsils be free of disease and the milk he ingests be uncontaminated, the presence of tubercle bacilli in the stomach contents is proof of the existence of a focus in the lungs. Unfortunately gastric lavage is unpleasant and the bacteriological examination is costly. The procedure should be undertaken only when the von Pirquet reaction is present and when there are symptoms suggestive of tuberculous infection of the lungs; by its means valuable information may be obtained.

Poulsen admits that many of these early lesions heal spontaneously, but he rightly remarks that no one is able to say with certainty whether such healing will occur or not; it is therefore in the best interests of the patient that a diagnosis should be made and appropriate treatment instituted. It might be added that any pulmonary tuberculous infection must have some deleterious effects on the

[1] *American Journal of Diseases of Children*, April, 1931.

child's well-being; it is fitting that all due efforts should be directed towards minimizing these effects. Furthermore, as Poulsen points out, children in whose stomach contents tubercle bacilli may be found, are infective; in the absence of a diagnosis by means of gastric lavage they would continue to be a menace to others in contact with them.

Poulsen supports his thesis by means of several case histories. In each instance he was unable to find any evidence of disease by means of clinical examination, and he did not consider the radiographic appearances were such as to warrant a diagnosis of pulmonary tuberculosis. He omits, however, to discuss in full the results of his percussion and auscultation of the chest, and there certainly is some doubt in regard to the X ray appearances. Though it is difficult to judge from brief reports and from greatly reduced reproductions of radiograms, we feel that in more than one of these cases it should not have been impossible to make a diagnosis of pulmonary tuberculosis by means of radiological examination. The question then arises, should diagnosis always be possible without the necessity of gastric lavage? Undoubtedly in the vast majority of instances the radiographic appearances are distinctive by the time tubercle bacilli appear in the sputum, but in rare cases, despite the employment of all ordinary measures, the diagnosis may remain in doubt. The discovery of tubercle bacilli in the gastric contents would then clinch the diagnosis and gastric lavage would be a justifiable procedure. More investigation is necessary before Poulsen's views in this important matter can be accepted in their entirety. Here is a field of research in the exploration of which the cooperation of clinician, radiologist and bacteriologist should most earnestly be sought.

Special Abstracts.

A PATHOLOGICAL STUDY OF TUBERCULOSIS.

The Medical Research Council of Great Britain, acting on the recommendation of the Tuberculosis Committee of the Council, has published a full account of comparative studies by Dr. H. H. Scott of tuberculosis in man and other animals.[1] The comparative method is of value in investigating the question of the portal of entry and subsequent paths of infection within the body. Since these two aspects of the study of tuberculosis are of the utmost importance from the preventive viewpoint, the following short account of Dr. Scott's work has been prepared for inclusion in this special issue of the journal. No attempt has been made to cover all the ground traversed by Dr. Scott; only those portions which appeared relevant have been mentioned.

Dr. Scott's material is drawn from *post mortem* examinations made by him in three hundred fatal cases of tuberculosis occurring among Chinese of the labouring class in Hong-Kong and from similar examinations of the series of wild animals dying of tuberculosis while in captivity in the Zoological Gardens, London.

[1] "Tuberculosis in Man and Lower Animals," by H. H. Scott, Medical Research Council of the Privy Council, Special Report Series, Number 149.

In the introduction to his report, Dr. Scott points out that the statement has been made that the bacillus of tuberculosis is ubiquitous and that no race is exempt. It would, he adds, be more correct to say that the bacillus finds a home wherever civilization penetrates and that, though certain races or communities are exempt, there is no proof that they are immune. If once the disease obtains a foothold in places where the natural morbidity rate is low, the mortality rate becomes very high. Domesticated animals, allowing for certain specific variations of susceptibility, occupy a position analogous to that of civilized or partly civilized communities and present a fairly heavy morbidity rate. Wild animals under natural conditions correspond to isolated races of human beings and it is highly improbable that tuberculosis exists among them. Wild animals in captivity form the counterpart of susceptible, but previously germ-free native races introduced into a civilized and tubercle-laden environment. When dealing with tuberculosis in animals, care must be taken to distinguish between a naturally acquired and an artificially produced infection. It is true that susceptibility is varied, but this variability must not be too greatly stressed in the natural state, because strict comparisons are not possible, since the different species are not kept under the same conditions.

Distribution of Lesions.

Part II of the report deals with the distribution of tuberculous lesions in man and other primates. This portion has been published in *The Journal of Pathology and Bacteriology* and is the joint work of Dr. Scott and Professor John Beattie. Dr. Scott points out that in this discussion the term primates does not include the New World monkeys. In these monkeys there is no true thoracic duct; the lymph from the abdomen and hind end of the body is collected into trunks which open into the venous system in the region of the junction of the renal veins with the inferior *vena cava*.

From the anatomical point of view it is shown that the topography of the lymphatic system of the animals examined (the types of animals are specified) does not differ in essentials from that of man. The arrangement of the terminal ducts in the apes and man is simpler than in some of the lower monkeys. The number of glands in a group varies directly with the size of the animals of a given genus. There is a basic pattern for each genus, and variations take place round this common pattern. A parietal thoracic gland of a given size appears to drain a definite superficial area. With increase in the size of the animal and corresponding increase of the surface area, the single gland is replaced by a group of glands. The arrangement in organs of a superficial plexus of lymphatic vessels not connected directly with the deep plexus of the same organ depends on the presence or absence of a definite capsule from a serous membrane. Superficial plexuses have been found only in those organs which are surrounded by a layer of serous membrane. The arrangement of plexuses and the connexions of glands can be determined only by injection experiments. Lymphatic vessels are formed in pathological adhesions in serous cavities. These lymphatic spaces are derived probably from a preexisting lymphatic plexus by an invasion of endothelial-lined vessels.

In the summary of his observations on the transmission of tuberculous material into distant regions, Dr. Scott states that the lymphatic system provides the principal channel for the transference of tubercle bacilli to distant parts of the body in early cases of tuberculosis in all primates. The order of glandular infection is the same in human tuberculosis as in tuberculosis of other primates. Tubercle bacilli can pass through lymphatic glands, especially when they become enlarged and inflamed. Normal glands retain the bacilli and in these circumstances tubercles are formed in the glands with breaking and caseation of the tissue.

Dr. Scott compares human tuberculosis in early life with tuberculosis in other primates. There are two distinct types of tuberculosis to be found both in man and other primates—the respiratory and the alimentary types. The former is a rapidly fatal infection causing death by general-

ized dissemination of bacilli by the lymphatics and blood vessels. The alimentary form (when it is primary) is less rapidly fatal. Alimentary tuberculosis may be secondary to a primary respiratory infection. The disease is transmitted to the gut by the swallowing of large numbers of bacilli in the sputum. Monkeys and apes never expectorate. In each type of the disease the dissemination of infection by the lymphatic system takes place in the same order in monkeys as in man. The proportion of alimentary to respiratory tuberculosis is probably much the same in all primates. Respiratory tuberculosis is identical in young human subjects and in other primates, but in later life in the former a modifying factor comes into play. Tuberculosis in early human life is the "natural" form of the disease. In other primates the modifying factor is absent and the adults are affected in the same way as the young animals. Dr. Scott gives details of the *post mortem* findings at autopsy of animals dead of tuberculosis and gives records of analogous conditions in the human subject. In several of these instances he discusses in detail the stages of dissemination and the route taken by the infecting organisms.

Tuberculosis in Man.

In the third part of his report, Dr. Scott discusses tuberculosis in man. He first of all refers to the several portals of entry and routes of extension of the infection.

Congenital Infection.

In regard to the question of congenital infection, Dr. Scott refers to the finding in the body of a baby of twenty-one days of a caseating focus and milia in one lung and numerous grey tubercles in the other. In a baby of twenty-four days he found a caseous focus in one lobe and grey tubercles disseminated throughout the remainder of both lungs; an even more advanced condition was found by him in an infant of twenty-nine days. In a child of seven weeks he found a caseated focus as large as a haricot bean in the lower lobe of the left lung, with miliary and small caseating tubercles throughout the right lung and the remainder of the left and infection of the diaphragmatic pleura on the left side. In another infant of seven weeks he found a caseous focus in the upper lobe of the right lung and cavitation the size of a cherry in the lowest lobe, miliary tubercles elsewhere in this lung and tuberculous bronchopneumonia in the lower lobe of the left lung and scattered milia in the upper lobe of the left lung and states that when he is faced with such conditions in children of so tender an age, he is practically forced to the conclusion that congenital tuberculosis does occur, and that infection has arisen *vid* the placenta of a tuberculous mother, or perhaps through the wall of the fœtal sac or that infection of the fœtus has occurred before the sac is formed. He refers to a report by Schluster of twelve cases in which there was evidence of tuberculosis at birth, and adds that the condition must be a very rare occurrence and even more rarely discovered. Schmorl and Birch-Hirschfeld, and Schmorl and Kockel have described cases in which the bacilli have been found in the placenta and fœtuses of tuberculous mothers. Friedmann reported two cases of infection *vid* the placenta. The fact that bacilli and microscopical tubercles are found in the placenta does not, according to Dr. Scott, necessarily imply infection of the fœtus, though this is probable. He suggests that possibly the bacilli and tubercles were confined to the maternal part of the placenta and that infection occurred during labour. His subsequent argument is as follows. The question of diathesis is fading more and more into the background, as facts are demanded in places of hypotheses. Infection may be accounted for without any necessity for relying on diathesis. The dominating factor is opportunity for infection; the greater the opportunity, the higher the rate of incidence. It is not sufficient to compare the death rate amongst the children of tuberculous parents with those of the healthy. Even were the former removed from the parents at birth and placed under the same conditions as the latter, though the position would be more comparable, the offspring of the tuberculous would still have been exposed to infection during the intrauterine period and to the debilitating effects of invalidism of their mothers in pregnancy. The children of tuberculous parents are not only exposed to danger of intrauterine infection, but they are more liable to infection in the early days of life, the dose of the infecting material is likely to be a large one and this dose is certain to be frequently repeated. Experimental work shows that size of dose and frequency of repetition are the main factors controlling infection in animals.

The Mouth and Nasal Passages.

In a vast majority of cases it is by the mouth or nasal passages that infection enters. Thence the bacilli may be inhaled or swallowed, or bacilli from a respiratory focus may pass up with the secretions, be coughed to the pharynx and be swallowed to set up intestinal disease secondarily, or the bacilli may pass without producing lesions in the mucosa and give rise to glandular infection, and thence by the lymph stream to the blood, and so an alimentary portal of entry may lead secondarily to pulmonary disease and later generalization.

Distribution of Lesions in Relation to the Portal of Entry.

In a long and detailed table, Dr. Scott describes the distribution of the lesions that were found in the three hundred *post mortem* examinations. The proportion of respiratory to alimentary infection is considerably higher in the tropics than is believed to occur in temperate zones. Amongst the 300 cases only 32 were definitely considered to be of alimentary origin (10·6%). Isolated primary intestinal tuberculosis was found in four cases only (1·3%), a contrast to the 25% recorded in temperate climates. In 209 instances (6·9%) the primary portal appeared to be respiratory. In four instances the findings indicated an almost simultaneous infection by both respiratory and alimentary tracts. In regard to the age mortality, 225 or 75% of the patients were below the age of ten years; examination of consecutive hundreds shows that this figure is a reasonably accurate estimate. There were 155 males and 145 females. Up to ten years the proportion of males to females was 4 : 5, between ten and twenty years the figures were equal, over twenty years the males were much in excess, namely, 3·6 : 1.

Analysis of Cases Occurring in Children.

Dr. Scott makes an analysis of the cases occurring in children according to the origin of infection. He divides them into groups in the same way as he does the tuberculosis of other primates. He gives typical instances of each type. Occasionally he uses the findings in animals to explain the appearances seen in children. Limitation of space prevents adequate description of this part of the work. It must suffice to state that he describes early cases of undoubted pulmonary origin; a later stage of undoubted pulmonary origin; a later stage of primary pulmonary disease, with commencing dissemination by the blood stream; cases of tuberculosis of pulmonary origin, with generalization by the blood vascular route; involvement of the alimentary tract secondary to pulmonary infection, but without generalization by the blood stream; final stages of primary pulmonary infection, the intestine being attacked by bacilli swallowed in sputum and the viscera infected by invasion of the systemic circulation. Among cases of intestinal origin he describes early primary intestinal tuberculosis, late primary intestinal infection, still later primary intestinal tuberculosis, with invasion of the blood stream.

General Aspects of the Disease in Children.

Canti, Albrecht, Ghon and others have made a special study of tuberculosis in children and have made certain generalizations. Dr. Scott takes these generalizations in turn and points out that in his series of 225 children several did not conform to these generalizations.

In the first place, it has been found that presence of tuberculous mediastinal glands was almost constantly indicative of a pulmonary focus. Among the 225 children there were 29, or nearly 13%, in which this did not obtain; that is, caseous tracheo-bronchial glands were present, although no focus could be found in the lungs. It is well

known that these glands may become involved secondarily to infection of the mesenteric glands. Since in 11 of the 29 evidence was in favour of the alimentary canal being the portal of entry, they may be dismissed from the total. Another might be excluded because it was likely that enlargement of the gland might have been caused by tuberculosis of the vertebral column. In all but two of the remaining 17 there was miliary infection of the lungs. The milia, though few in twelve instances, were more numerous in others and were generally and not focally distributed. In two the lungs presented no tuberculous infection.

In the second place it has been found that tuberculous mediastinal glands, when present, bear a close relation to the focus in the lung. In a few of the cases the gland and the focus were not found to correspond. Details are given; the lesion was found in one lung and glandular involvement, or more pronounced glandular involvement, in the other. It is well known that communication may be free between the glands on one side and those on the other, and this, it is suggested, may account for the passage of infection in some of these cases.

Thirdly, the investigators previously mentioned have laid stress on the frequent singleness of the lung focus. Dr. Scott points out that it is not always easy to determine what is meant by a focus. A small focus may be the result of a broken down focus, or may be caused by the fusion of adjacent patches of tuberculous bronchopneumonia. Dr. Scott has not regarded as foci such conditions as localized breaking down to cavitation if there is fairly general tuberculous bronchopneumonia; but when there is a focal caseous mass or cavity more or less isolated or with milia only in the vicinity, so that it may be inferred that the breaking down has taken place in a previously existing focus, this has been included. Of the 225 children in the series there were 123 with focal pulmonary tuberculosis. Of these, 85 presented but a single focus (69·1%), 29 had two foci (23·5%) and nine had three foci or more.

In the fourth place it has been stated that when a focus is present in the lung, tuberculous mediastinal glands are constantly found. There are exceptions. It may be contended that in some instances death takes place before there is time for glandular involvement to arise. Dr. Scott finds a certain amount of support for this suggestion. At the same time he gives details of three cases in which this explanation will not hold.

In the fifth place it is said that a lung focus is almost constantly absent when the portal of entry appears elsewhere. Dr. Scott cannot consider this apart from a sixth point: the almost constant absence of evidence that the portal of entry might be elsewhere when a lung focus is present. In this regard it is necessary to exclude all cases in which dual infection might be present. Dr. Scott refers to three cases in the series in which there seemed to be no doubt that the primary portal of entry was alimentary, but focal lesions were present in the lungs.

"Modified" Tuberculosis of Adults.

By "natural" tuberculosis is implied a disease caused by the tubercle bacillus in an animal or a race not immunized in any way racially against the toxin of the bacillus. "Modified" tuberculosis is a disease caused by the tubercle bacillus in man alone and characterized by fibroid changes around the lesion and by a chronicity in its course; it is mainly local in its spread; that is, it spreads by contiguity rather than by the lymph stream. The modified disease is thus usually a secondary infection either by the lighting up of an old focus or by a reinfection. Dr. Scott points out that the tuberculosis in children discussed by him is "natural" tuberculosis. Of his 300 autopsies only 75 were on bodies of persons over the age of ten years, and of these only 65 could be strictly regarded as adults.

While in only one of the 225 children was an old healed or shut-off focus found, eleven such foci were found among the 65 adults. Of these eleven, there were six with calcified deposits, three with definite cicatrices and two with fibroid phthisis. One of the two last mentioned had a calcareous focus in one lung and also an old subapical scar in the other. Death in most instances was due to phthisis and a tuberculous bronchopneumonia, active and of more recent date than the old focus; in other words, a reinfection.

It was the exception rather than the rule for corresponding glandular involvement to occur with lung tuberculosis in adults. Among the 65 adult cases there were twelve in which there was extensive pulmonary tuberculosis, in the majority with cavities, in others with a definite caseous focus, but no signs of any enlargement of the corresponding glands.

In three cases death was due to a large pulmonary hæmorrhage. Not one instance was found among the children. It would seem that this occurs only in the modified disease.

There was a far greater preponderance of cases with a terminal meningitis amongst adults than amongst the young. There were 24 cases in which a terminal meningeal tuberculosis was found apart from a general dissemination of tubercles. One was in an adult of sixteen years; ten were amongst the 225 under ten years of age; 13 were amongst the 65 adults.

Tuberculous Septicæmia.

Dr. Scott found it not a very rare experience to meet with instances in which there were indications of an invasion of the blood stream with fairly widespread dissemination of miliary tubercles, but in which careful search failed to reveal any focal lesion or evidence as to the primary portal of entry. In some of these instances there was a swollen gland which on section revealed one or two points of caseation. These could hardly be looked on as a focus for setting up generalized infection. In the same way by which an infected mesenteric gland is an indication of the alimentary portal of entry of the bacilli, even though no demonstrable affection of the gut can be made out, so may a mediastinal gland become infected without it being possible to demonstrate bacilli in the air passages. In some sections of a necrotic focus an open vessel might be seen traversing the tuberculous focus, still containing unaltered blood corpuscles and therefore still active in carrying on the circulation. Dr. Scott regards it as quite possible that in these circumstances the constant entrance of bacilli, even though in small numbers at a time, may bring about generalization; he suggests that this is the probable explanation of some of the cases which have been designated tuberculous septicæmia.

THE BACILLE CALMETTE-GUÉRIN.

THE *Bacille Calmette-Guérin* is a tubercle bacillus of an attenuated strain and is said to be non-pathogenic. Claims have been made that by its administration persons may be immunized against tuberculosis. Its discovery, therefore, was hailed with delight by health authorities in many parts of the world. In England and some other countries, however, it was received with some scepticism. Though in France *Bacille Calmette-Guérin* vaccine has been enthusiastically employed for several years, British opinion has remained conservative pending further experimental evidence of the harmlessness of this bacillus and its value as an immunizing agent. In other parts of the world there have been conflicting reports. In view of the existing doubt and confusion a study of the results of British scientific experiment should be of value. Recently the Medical Research Council has published a brochure by Dr. A. Stanley Griffith, entitled "Studies of Protection Against Tuberculosis: Results with B.C.G. Vaccine in Monkeys."[1]

INTRODUCTION.

Dr. Griffith remarks that in 1921 Weill-Hallé showed that large quantities of a culture of *Bacille Calmette-Guérin*

[1] "Studies of Protection Against Tuberculosis: Results with B.C.G. Vaccine in Monkeys," by A. Stanley Griffith, Medical Research Council of the Privy Council, Special Report Series Number 152.

could be ingested by young infants without any apparent ill effect. Weill-Hallé and Turpin, from July, 1922, to December, 1925, inoculated 317 infants, with favourable results.

In 1923, at the instigation of Calmette, Wilbert undertook an investigation of the effects of the administration of *Bacille Calmette-Guérin* vaccine to monkeys in French Guinea. Calmette insisted that, as monkeys are so highly susceptible to tuberculosis, the work must be carried out in a country where they could be kept in a good state of health and where risk of natural infection with human tubercle bacilli was at a minimum. It was first of all ascertained that *Bacille Calmette-Guérin* vaccine was harmless and that a suitable dose was 50 milligrammes administered subcutaneously or 50 milligrammes on each of five alternate days by the mouth. The bacilli used for injection, when administered in a dose of 0·00001 milligramme subcutaneously or 0·001 milligramme orally, had been proved to cause tuberculosis fatal to monkeys in three to six months.

Fifteen chimpanzees and fifty-six monkeys were used in the experiments. None of the vaccinated animals became affected with tuberculosis, though in one instance a chimpanzee was inoculated subcutaneously with 0·1 milligramme of a culture of bovine tubercle bacilli. Of the controls, all those who did not die of some acute illness, died of tuberculosis. A report was published in 1925.

A committee was appointed by the Medical Research Council to consider the question of the employment of *Bacille Calmette-Guérin* vaccine. Though the members of this committee were impressed with Wilbert's results, they concluded that confirmation was desirable, and therefore, in 1926, they instructed Dr. Griffitn to conduct experiments. During the period in which he was engaged on this work, Tzechnovitzer, Schlossmann, Gerlach and Kraus, Kirchner and Schneider, Kalbfleisch and Nohlen, and L. Lange also made investigations. Gerlach and Kraus, though their observations were made over a period of three months only, concluded that the administration of *Bacille Calmette-Guérin* vaccine could produce in monkeys an immunity against experimental tuberculosis. In some of the experiments of the other workers named, some of the vaccinated animals did not contract tuberculosis, but in most instances there was no evidence that immunity resulted from the administration of *Bacille Calmette-Guérin* vaccine.

The species of animal used in Dr. Griffith's experiments was *Macacus rhesus*. The monkeys were imported to England from India. Notwithstanding Calmette's view that on account of the liability of monkeys to spontaneous tuberculous infection, experiments should not be conducted in Europe, Dr. Griffith maintains that, if care be taken and if the animals be specially selected in India and obtained immediately on their arrival in England, such complication is a rarity. Still, the possibility must be borne in mind. Therefore, throughout his work Dr. Griffith always employed tubercle bacilli of strains dissimilar to those likely to be encountered naturally, made careful *post mortem* examinations in order to determine the seat of the primary lesion, and compared cultures of organisms from the lesions with cultures of the organisms used in the experiments.

The strain of *Bacille Calmette-Guérin* employed was obtained from Calmette in 1921.

Virulence of Bacille Calmette-Guérin.

One monkey inoculated intravenously with 50 milligrammes and one with 10 milligrammes of *Bacille Calmette-Guérin* vaccine died in 13 days and 32 days respectively "as a direct result of the irritant and toxic effects of the bacilli." In both instances there were pneumonic patches in the lungs and *Bacille Calmette-Guérin* was obtained from the organs; in one instance small tubercles were present. A third monkey was given an intravenous injection of one milligramme, followed in 79 days by a second injection of two milligrammes. It died 21 days later of non-tuberculous pneumonia. There was no evidence of tuberculosis, and *Bacille Calmette-Guérin* was not recovered from the organs. Other animals were given large doses intratracheally, subcutaneously, orally, and by instillation into the conjunctival sac. Griffith concludes that the *Bacille Calmette-Guérin* does not cause progressive tuberculosis and that it may remain alive in the lymphatic glands for six months without provoking characteristic tuberculous lesions, but that, when it is administered in large doses intravenously or intratracheally, it causes tuberculous pneumonia and even the development of definite tubercles. The lesions in such instances, however, are limited to the lungs and have no tendency to progress.

Absorption of Bacille Calmette-Guérin from the Alimentary Tract.

Four monkeys were fed with quantities of *Bacille Calmette-Guérin* vaccine ranging from 250 milligrammes to 1,030 milligrammes, a fifth was given instillations into the conjunctival sac (the dose is not mentioned). Tubes of culture medium were later inoculated from various lymphatic glands, spleen and liver. In one instance the result was indeterminate owing to contamination by secondary organisms. *Bacille Calmette-Guérin* was cultured from the retropharyngeal glands of a monkey fed with 300 milligrammes of vaccine; from cervical and mesenteric glands of a monkey fed with 420 milligrammes; from cervical, mesenteric, ileo-caecal and colic glands and spleen of a monkey fed with 1,030 milligrammes; and from cervical and mesenteric glands of the monkey whose eye had been inoculated. Griffith concludes that *Bacille Calmette-Guérin*, given by mouth, can pass through the wall of the alimentary tract into adjacent lymphatic glands, and also into the blood stream, but that the organism is not absorbed in numbers likely to be sufficient to produce a demonstrable immunity, unless the vaccine be administered in larger doses and over a longer period than recommended by Calmette.

Immunity Experiments.

In a series of five experiments Dr. Griffith endeavoured to ascertain whether or not immunity could result from the administration of *Bacille Calmette-Guérin* vaccine. In several experiments he inoculated the animals with an infective dose of tubercle bacilli by the instillation of an emulsion into the conjunctival sac; in other instances the animals were exposed to infection by contact with other tuberculous monkeys. In a sixth experiment he employed subcutaneous inoculation. This latter he regarded as too severe a test; the *rhesus* monkey is very highly susceptible to the action of tubercle bacilli in the tissues. One monkey died of generalized tuberculosis within 49 days of subcutaneous inoculation with a dose of 0·00001 milligramme of human tubercle bacilli. The method of infection by feeding has certain disadvantages. There is the possibility that, if a minute dose be given, the bacilli may all be passed out with the fæces. If sufficiently large doses be administered to insure infection, there are wide variations in the extent and course of the resulting disease, for there are differences in the rates of absorption from the alimentary tract. The monkey may discard some of the food containing the bacilli; if, to obviate this, the emulsion be placed in the mouth by means of a pipette, there is a danger of direct infection of the lungs by insufflation. There are no such objections to the method of infection by instillation into the conjunctival sac. When this method is used, the local signs of infection are conjunctivitis and enlarged cervical lymphatic glands. These afford a ready means of ascertaining whether the inoculation has resulted in the development of disease and of observing the time of appearance and the progress of the lesions. The method has advantages over infection by contact, for it permits of infection by a known dose, by a definite route and at a fixed time. Furthermore, on account of their distribution, the lesions of spontaneous tuberculosis cannot readily be mistaken for the lesions resulting from instillation of bacilli into the conjunctival sac.

Inoculation by Instillation and Contact.

First Experiment.

In the first experiment the dose of tubercle bacilli was 0·01 milligramme. Two monkeys were vaccinated by

mouth with 500 milligrammes of *Bacille Calmette-Guérin* vaccine, two subcutaneously with 50 milligrammes; two were used as controls. About two months after vaccination the two monkeys which had received the vaccine subcutaneously reacted to the tuberculin test, the others did not. One control monkey died 74 days after the instillation of tubercle bacilli into his conjunctival sac, but the lesions did not follow the expected arrangement, and Dr. Griffith concludes that the animal had already been infected naturally at the time of the inoculation. The other control was killed 114 days after inoculation and was found to be affected with tuberculous adenitis and generalized tuberculosis. All four vaccinated monkeys developed generalized tuberculosis, but those which had been vaccinated by subcutaneous injection were not affected so early as were the controls or those which had been vaccinated by the mouth. The results of this experiment, then, did not confirm the findings of Wilbert.

Calmette, in a private communication to Dr. Griffith, remarked concerning this experiment that the instillation of 0·01 milligramme of bacilli was too severe a test and that *"Il n'y a pas de prémunition qui puisse tenir en face d'une infection aussi massive."* Dr. Griffith does not agree that the dose was overwhelming or so large as to prevent an immunized animal from giving evidence of the possession of some powers of resistance. He remarks that a dose could not be regarded as *"massive"* which did not cause the death of an unvaccinated monkey within four months. Finally, natural disease may result in the development of power to resist infection by this dose, for, as mentioned above, an infected control monkey did not develop experimental tuberculosis.

The Second Experiment.

In the second experiment an intravenous injection of 1·0 milligramme of *Bacille Calmette-Guérin* vaccine was given to one monkey; one was fed with 950 milligrammes and one was vaccinated subcutaneously with a dose of 50 milligrammes. One hundred and eight days after vaccination of the last mentioned monkey an abscess at the site of injection was still discharging pus containing acid-fast bacilli. One monkey was used as a control.

A dose of 0·001 milligramme of a culture of tubercle bacilli was instilled into the conjunctival sac of each of the monkeys. The control animal died 38 days after inoculation. There were no pronounced tuberculous lesions, but tubercle bacilli were observed in a smear from the submaxillary glands, and injections of emulsions of tissue from spleen and submaxillary glands proved fatal to guinea-pigs. The other three monkeys died of generalized tuberculosis, 176, 193 and 236 days respectively after inoculation. Dr. Griffith remarks that, as the control monkey died prematurely, it cannot be decided whether the relatively mild type and protracted course of the disease in the treated animals were due to increased resistance or some factor concerned with the bacilli.

The Third Experiment.

In the third experiment five monkeys were vaccinated by feeding with *Bacille Calmette-Guérin* vaccine over a period of several months. The total doses varied from 110 milligrammes to 805 milligrammes. Three controls were employed. All were inoculated by the instillation of 0·001 milligramme of bovine tubercle bacilli into the conjunctival sac. One control monkey was apparently suffering from a natural tuberculous infection at the time of inoculation, for, 324 days later, its lungs were found to be infected with human tubercle bacilli and bovine bacilli were not found in its tissues. One of the vaccinated monkeys suffered from a natural infection, but the bacilli used in the test also invaded its tissues and caused lesions. The other vaccinated animals all became affected with severe tuberculosis produced by the organisms used in the inoculation, but the disease was more protracted in them than in the controls.

The Fourth Experiment.

One monkey vaccinated by mouth, one control and an experimentally infected animal were used in the fourth experiment. They were placed in a single cage. The infected animal died of generalized tuberculosis 56 days after the commencement of the experiment; the control monkey died 73 days after the day on which it was first exposed to infection, of tuberculosis of a human type, which had probably been contracted before the commencement of the experiment; the vaccinated monkey died of generalized tuberculosis 88 days after the commencement of the experiment. The bacilli in the experimentally infected animal were of the same strain as in the lesions of the vaccinated animal. Dr. Griffith concludes that infection was acquired by contact.

The Fifth Experiment.

In the fifth experiment the monkeys were divided into three groups. Four vaccinated monkeys and two controls were inoculated by instillation in the conjunctival sac with 0·001 milligramme of a culture of bovine tubercle bacilli. One monkey did not apparently become infected as a result of the inoculation, but, despite further vaccination by feeding, contracted infection by inhalation. The other three monkeys became infected, despite that one of them jerked its head suddenly when the instillation was being performed and received probably little more than one-quarter of the dose.

Two vaccinated monkeys and two controls were inoculated by conjunctival instillation of 0·01 milligramme of a culture of bovine tubercle bacilli. One, vaccinated intratracheally and intravenously, died of diarrhœa after 36 days; no tuberculous lesions were found, but tubercle bacilli were isolated from submaxillary and mesenteric glands. The other monkeys contracted tuberculosis.

Five vaccinated monkeys and two controls were placed in the same cage with two inoculated, unvaccinated monkeys. The two inoculated monkeys died of tuberculosis 97 and 111 days from the commencement of the experiment. Three of the vaccinated monkeys and one control monkey escaped infection; the others developed respiratory tuberculosis.

Inoculation by Subcutaneous Injection.

The Sixth Experiment.

In the sixth experiment one monkey vaccinated with *Bacille Calmette-Guérin* by intravenous injection, two by intratracheal injection and two by subcutaneous injection were inoculated subcutaneously with 0·0001 milligramme of a culture of bovine tubercle bacilli which had lost some virulence after two and a half years' cultivation. Two control monkeys were inoculated and died of generalized tuberculosis 65 and 78 days later. Two of the vaccinated monkeys died prematurely; both were infected. Two which had been vaccinated subcutaneously died of generalized tuberculosis 69 and 73 days after inoculation. The remaining animal, which had been vaccinated intratracheally, died 112 days after inoculation.

SUMMARY.

Instillation in the Conjunctival Sac.

Of ten monkeys vaccinated by feeding and inoculated by instillation of emulsions of tubercle bacilli in the conjunctival sac, three died of generalized tuberculosis differing in no way from that observed in the controls; three died of generalized tuberculosis after longer periods than were occupied by the course of the disease in the controls; one received less than the test dose and lived longer than the controls; one, killed after the lapse of 84 days, was found to be affected with less severe internal disease than the controls; one died of pulmonary tuberculosis contracted naturally; one escaped infection by the instilled bacilli but later contracted pulmonary tuberculosis by inhalation.

Of four monkeys vaccinated subcutaneously and inoculated by instillation of emulsions of tubercle bacilli in the conjunctival sac, one became affected with tuberculosis of a much milder type than that of the controls; one contracted disease equal in extent and severity to that of the controls; two were affected a little less severely than the controls.

Infection by Contact.

In two experiments three of six vaccinated monkeys and one of two controls contracted tuberculosis from experimentally infected animals with which they had been placed in contact.

Infection by Subcutaneous Inoculation.

Of five vaccinated monkeys tested by subcutaneous inoculation, two died prematurely; two died of generalized tuberculosis after a period about equal to the duration of the disease of the controls; the fifth lived a month longer than the controls, but died of generalized tuberculosis.

CONCLUSIONS.

Dr. Griffith remarks that the results of his experiments do not confirm Wilbert's conclusion that monkeys can be completely immunized against tuberculosis by vaccination with *Bacille Calmette-Guérin*. In some instances the disease experimentally produced in vaccinated monkeys was more protracted in its course than was the disease of the controls, but he is unable to say whether this was due to increased resistance or to some other circumstance, such as the age of the monkey, natural resistance, or lowered virulence of the bacilli. If it be accepted that an acquired resistance was induced in these instances, it is apparent that the administration of *Bacille Calmette-Guérin* vaccine "in the ways recommended by Calmette can produce, irregularly and inconstantly, at most only a low grade of relative immunity in the monkey."

Obituary.

ERIC IVO LOWTHER GRAVES.

WE regret to announce the death of Dr. Eric Ivo Lowther Graves, which occurred on July 23, 1931, at Brunswick, Victoria.

Corrigendum.

AT the foot of the letter from Dr. Arthur Murphy, published in the issue of July 25, 1931, Dr. Murphy's hospital appointment was incorrectly stated. The correct appointment is Honorary Surgeon, Ear, Nose and Throat Department, Mater Misericordiæ Children's Hospital, Brisbane.

Diary for the Month.

AUG. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 13.—New South Wales Branch B.M.A.: Clinical Meeting.
AUG. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 26.—Victorian Branch, B.M.A.: Council.
AUG. 27.—South Australian Branch, B.M.A.: Branch.
AUG. 27.—New South Wales Branch, B.M.A.: Branch.
AUG. 28.—Queensland Branch, B.M.A.: Council.
SEPT. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 2.—Victorian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.
NATIONAL ASSOCIATION FOR THE PREVENTION AND CURE OF CONSUMPTION, SYDNEY, NEW SOUTH WALES: Honorary Physician.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Clinical Assistant to Surgical Out-Door Department.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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